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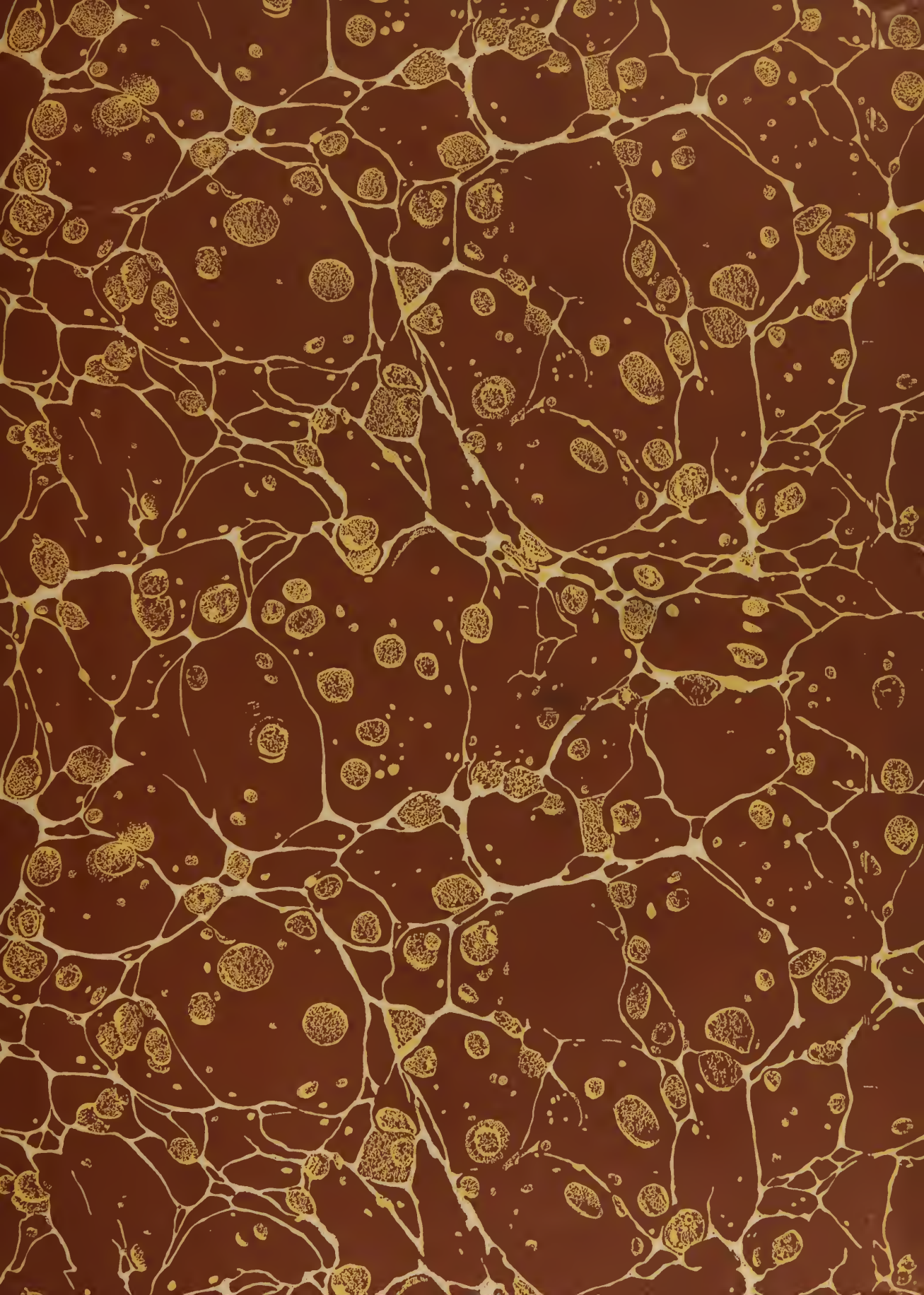
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The SPECIAL PATHOLOGY and TREATMENT

—OF—

THE ORGANIC

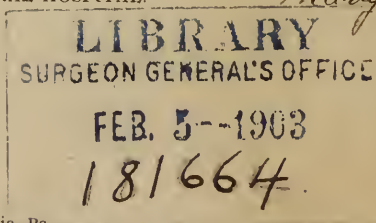
Diseases of the Stomach,

—BY—

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presented by the author.
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IN TWO PARTS.

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TO MY GOOD FATHER,

MR. JOHN HEMMETER,

AND MY FATHERLY FRIEND,

MR. CHARLES HILGENBERG,

THIS VOLUME IS GRATEFULLY DEDICATED.



PREFACE.

IN AN ADDRESS before the Medical and Chirurgical State Faculty of Maryland in April 1896, Professor Da Costa in speaking of the manner in which Medical Libraries build up and increase, said that "Books attract books, and as a rule, any new work in any particular class has a striking family resemblance to those already published."

If this new contribution to the Pathology and Treatment of Organic Diseases of the Stomach does not conform to Da Costa's generalization, it is not because of any premeditated plan to make it different from other works on the same subject, but because a number of entirely new methods of diagnosis have entered into it, and, because an effort has been made to do justice to the work of American Clinicians on this special department, which foreign books, as a rule omit, except in a few instances where the American contribution is so indispensable that they can not consistently avoid reference to it.

When the printing of this book was begun, there was no work of American origin on this subject. Since then the volume by Max Einhorn has appeared as the pioneer.

This small volume is printed from the advance sheets of a larger and more exhaustive work to be published in October, 1897 and has been compiled for the benefit of the author's class at the Baltimore Medical College.

We already have a large number of eminently qualified and versatile clinicians, men with acute observing powers and analytical minds who have worked in this interesting field. The names of Austin Flint, Pepper, Osler and Delafield are as well known in this department in our country, as those of Kussmaul, Senator, Leube and Ewald in Germany.


The work of Einhorn will mark an epoch in the history of gastro enterology in this country, ably supplemented and supported by the researches of D. D. Stewart, Charles G. Stockton, Allen Jones, Julius Friedenwald, Francis P. Kinnicut, Charles E. Simon and other gifted experimenters and observers.

The Surgery of the Alimentary tract has many very creditable representatives in our country among whom may be mentioned, Robert F. Weir, N. Senn, McBurney, Roswell Park, F. Lang, R. Abbe, W. Meyer, Murphy, Bull, Maurice H. Richardson, Gerster, John M. T. Finney. The literary and practical contributions of a number of these men has reached a classic standard and compelled foreign admiration.

The Physiological Chemistry of Digestion and Internal Secretion has received the benefit of the work of Bowditch, Chittenden, Howell, Vaughn, Adami, Able and others, and Dietetics has its versatile representative in Gilman Thompson.

To Dr. Edward L. Whitney, my associate, it becomes my pleasant duty to express thanks for the manner in which he has written the chemical section of Part first and much kind assistance throughout the work.

Pathology has its men now universally acknowledged for the integrity and dignity of their work in our esteemed teachers Welch and Councilman. Already an American School of Pathology is forming with these men, and Prudden, Flexner and others. But in the special Pathology of the Digestive organs, the workers are few, a very creditable beginning however, has been made; the foundation is an honor to the prospective builders, — The land to be explored is exceedingly large in its extent,— *“The harvest is plenteous, but the laborers are few.”*



“Heard are the voices,
Heard are the sages,
The worlds and the ages;
Chose well, your choice is
Brief and yet endless.”

“Here eyes do regard you
In eternity’s stillness,
Here is all fullness,
Ye brave to reward you,
Work and despair not.”

(Goethe)

PART FIRST.

ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE ORGANS.

THE METHODS AND TECHNICS OF DIAGNOSIS.

LECTURE I.

THE ANATOMY OF THE STOMACH AND INTESTINES.

(I) THE STOMACH.

THE STOMACH is the dilated, sac like portion of the digestive tract, between the oesophagus and small intestine; one can distinguish a lower convex arch, the greater curvature which is directed toward the left and downwards, and an upper concave arch, the lesser curvature, which is directed toward the right and upwards; the broad left end of the greater curvature is called the fundus, whose size varies according to age. Between this and the lesser curvature is situated the cardia, this is the boundary between the oesophagus and the stomach, and while it is not marked on the outside of the organ, there is a distinct limiting line internally on the mucous membrane, which is caused by a change in the structure of the epithelial lining. It is now well known that at this point the arrangement of the muscular fibers and veins is also different from that in the oesophagus.

The location of the cardia in the adult is at the twelfth thoracic vertebra; at about the height of the bifurcation of the bronchi; the spiral curving of the oesophagus around the aorta begins, by executing this curve, the convexity of which is towards the right, the oesophagus gets to the left side of the aorta, and passes through the diaphragm in the foramen oesophageum near the spinal column.

The stomach becomes narrower from the fundus towards the pylorus; near the pylorus there is a constriction caused by a ring-like formation of muscular tissue, which corresponds to the pyloric valve; the muscular tissue is covered internally by the gastric muc-

ous membrane, the latter forming the pyloric valve; the opening of this valve is of varying diameter; the part of the stomach preceding the pylorus is called the antrum pyloricum, and is frequently separated from the greater curvature by an indentation or depression, this antrum may be elongated so as to assume resemblance to the intestine which is frequently the case in the female sex.

On the anterior and posterior wall of the stomach running along between the muscular and serous coat of the organ, are two band-like stripes, consisting of elastic, smooth, muscular fibers, these are the pyloric ligaments.

The size of the stomach depends upon the age, sex, individuality and degree of its distention. In the female sex it is generally small and more slender; the long axis extends from twenty-five to thirty-five c. m. The greatest vertical measurement at the cardia is fifteen c. m., and the greatest straight diameter is eleven to twelve; the smallest at the antrum pyloricum is from three to four centi meters.

The capacity varies considerably; Ewald considers that sixteen to seventeen hundred c. c. is the normal limit; $\frac{3}{4}$ of the stomach belong to the left body half, and $\frac{1}{4}$ to the right. The cardia is located behind the median edge of the fifth and sixth ribs. The fundus, the largest part of the body, is in the left hypochondrium; the rest with the pyloric part is in the epigastrium. The pylorus lies in the right half of the body, but occasionally changes to the middle line at the level of the seventh and eighth ribs, in a line with the ensiform cartilage. The lesser curvature runs along to the left and near to the spinal column. The vaulting dome of the fundus, which applies itself to the concavity of the diaphragm is the highest point; the deepest point of the stomach is in the greater curvature; in the inferior $\frac{1}{2}$ of a straight line connecting the ensiform cartilage with the umbilicus; both the highest and lowest part of the stomach are moved about according to the level of the diaphragm and the distention of the stomach; In an empty condition, the stomach is withdrawn into the upper portion of the abdomen, but when filled it extends in all directions, but mostly in the direction of its long axis, from the left above to right downwards, in a state of moderate distention, about forty c. m. of its anterior wall come in contact with the inner surface of the anterior abdominal wall.

The diaphragm covers the fundus and the largest part of the

left segment, while the left lobe of the liver up to the sulcus interlobularis covers over the smaller part, that is, the lesser curvature and the pyloric portion. From this fact arises the difficulty to palpate tumors in the latter place, which is impossible, except when in gastropsis, descent of the stomach moves it away from the liver; in the state of expansion or dilatation the stomach moves out from behind the liver, but the lesser curvature cannot change its location to any considerable extent, and the change of location of the whole stomach caused by filling, is produced almost exclusively by an extension of the greater curvature.

The pancreas extends along the posterior wall of the stomach; at the upper edge of the pancreas are the lienalis artery and vein. The transvers colon runs along the greater curvature, and its left flexure fills the remaining space in the left hypochondrium. The location of the stomach is fixed by a ligamentous attachment of the cardia, and the pylorus, and also by a number of suspensory ligaments, which are all formations of the peritoneum; some authors say that the stomach is supported in this position by intra-abdominal pressure; the experiments of Moritz of Munich, and myself have proven that intra-abdominal pressure adds nothing to the support of the stomach. The gastro-phrenic ligament which towards the right passes into the lesser omentum, and towards the left extends into the phrenico-lienal ligament surrounds and embraces the cardia; the cardia sits lower than the fundus, and its situation corresponds to the upper end of the sixth and seventh costal cartilages, or to the level of the ninth thoracic vertebra; this part of the stomach is therefore moved to the left of the middle line, and next to the spinal column; at about the level of the twelfth thoracic and first lumbar vertebræ, here it is fixed to the lumbar part of the diaphragm.

The greater omentum arises from the large curvature. The posterior fold of this omentum forms the meso-colon transversum;

— This is the reason why changes of location in the greater omentum (hernia and inflammatory adhesions) can produce traction of the stomach, as the stomach is really attached only at the cardia, and the pylorus adheres to the posterior abdominal wall, by the descending portion of the duodenum; the stomach is capable of being moved about, not so much in its entirety as in its parts (the great curvature for instance). The stomach has a complete peritoneal covering, which consists of an an-

terior and posterior layer, these layers uniting at the two curvatures of the stomach to form the lesser and greater omentum; between these two layers a space is left for the blood and lymph vessels of the stomach.

The muscular stratum contains three kinds of fibers longitudinal, transverse, and oblique. The longitudinal layer of muscular fibers, a continuation of those of the oesophagus, presents a denser arrangement at the lesser curvature than at the greater, and forms the ligamenta pylorica at the pyloric part, which are bands of muscular fibers, expanded and broadened out, not ligaments in the real sense of the word.

The circular layer of muscular fibers is placed internally to the longitudinal layer, the fibers of which are crossed at right angles. The fibers of this circular layer are placed around the stomach in a ring, or belt like manner; at the pylorus it shows a local thickening of the muscle rings, the sphincter pylori; a fold of the mucosa to the innermost side of this sphincter constitutes the pyloric valve. The longitudinal fibers also have a part in the formation of the sphincter, for whilst the superficial layer of longitudinal fibers passes on over the pyloric sphincter into the duo-denum, the deeper longitudinal fibers enter the pyloric valve and encircle and grasp the circular fibers in a loop like manner (*Dilator Pylori*—Rudinger). The cardia has no special sphincter, but the oblique fibers, cross and decussate at the periphery of this portion. As the sphincter pylori is contracted during digestion, gas and liquids can readily escape through the cardia. The oblique fibers are limited chiefly to the cardiac end of the stomach, where they are disposed as a thick uniform layer, some passing obliquely from left to right, others from right to left around the cardiac orifice. The submucosa, or cellular coat of the stomach consists of a loose filamentous, areolar tissue, and loosely binds the mucosa to the muscular layers.

The most important, and interesting is the mucous layer, or mucous membrane proper of the stomach; it is a thick layer with a smooth soft velvety surface. During infancy, and immediately after death it is of a pinkish tinge, but in adult life and old age, it becomes of a pale straw or ash grey color; at the pylorus it is much thicker than at the cardia. During the contracted state of the organ, it is thrown into numerous plaits or rugae, which, for the most part,

has a longitudinal direction, and are most marked towards the lesser end of the stomach, and along the greater curvature; these folds are entirely obliterated when the organ becomes distended.

STRUCTURE OF THE MUCOUS MEMBRANE.

When examined with a lens, the inner surface of the mucous membrane presents a peculiar honey-comb appearance, from being covered with small shallow depressions or alveoli, of a polygonal or hexagonal form, which vary from one one-hundredth to one three-hundred and fiftieth of an inch in diameter, and are separated by slightly elevated ridges. In the bottom of the alveoli are seen the orifices of minute tubes, the gastric follicles, which are situated perpendicularly side by side, in the entire substance of the mucous membrane; they are short and simple, tubular in character, toward the cardia; but at the pyloric end, they are longer, more thickly set convoluted, and terminate in dilated saccular extremities, or are subdivided into from two to six tubular branches.

Watney has pointed out that these convoluted, or coiled tubes form the transition from the simple tubular follicles, to the convoluted glands of Brunner, which lie immediately below the pylorus. The gastric follicles are composed of a homogeneous basement membrane, lined upon its free surface by a layer of cells, which differ in their character in different parts of the stomach. Towards the pylorus these tubes are lined throughout by columnar epithelium; they are termed the mucous glands, and are supposed to secrete the gastric mucus. In other parts of the organ, the deep parts of each tube is filled with nuclei, and a mass of granules; above these are a mass of nucleated cells, the upper fourth of the tube being lined by columnar epithelium: These are called the peptic glands, and are the supposed agents in the secretion of gastric juice.

Simple follicles are found in greater or less number over the entire surface of the mucous membrane; they are most numerous near the pyloric end of the stomach, and are especially distinct in early life. The epithelium lining the mucous membrane of the stomach and its alveoli is of the columnar variety.

VESSELS AND NERVES.

The arteries supplying the stomach are, the coronaria ventriculi, the pyloric and right gastro-epiploic branches of the hepatic, the left

gastro-epiploic and vasa brevia from the splenic. They supply the muscular coat, ramify in the submucous coat, and are finally distributed to the mucous membrane. The arrangement of the vessels in the mucous membrane is some-what peculiar. The arteries break up at the base of the gastric tubules into a plexus of fine capillaries which run upwards, between the tubules, anastomosing with each other, and ending in a plexus of large capillaries, which surround the mouth of the tubes, and also form hexagonal meshes around the alveoli; From these latter the veins arise, which pursue a straight course back to the submucous tissue, between the tubules, to terminate in the splenic and portal veins.

The lymphatics are abundant and may be classed into a superficial and deep set, which pass through the lymphatic glands found along the two curvatures. The nerves are supplied from the right and left pneumogastric, and numerous branches from the abdominal sympathetics.

Usually 4 to 6 gland openings are found at the base of each follicle. According to Sappey there are 5,000,000 of these glands in the organ, for which reason the gastric mucosa may justly be considered a continuous gland that is spread out into a flat surface. —(Hyrtl and Luschka). The gland tubules are as long as the entire thickness of the mucosa, and their sac-like and branched bases extend into the muscularis mucosa, the action of these muscular fibers assist in the evacuation of the tubules during digestion; These ends of the tubules extending into the muscular layer are usually branched.

The cylindrical epithelium of the surface of the stomach which is separated from the pavement epithelial lining of the oesophagus by a distinct but irregular line at the beginning of the cardia, — is found also in the beginning of the tubules, but only extends downward to $\frac{1}{4}$ or $\frac{1}{3}$ their length; from this point downward they are no longer observed, but two different kinds of cells are now seen to line the glandular tubule. One of the variety of lining cells of the gland tubules is located toward the axis of the tube, and is known as the adelomorphous cells of Rollet, because they show no cell contours or outline in the fresh state, called by Haidenhayn chief cells, they have a cubical, cylindrical form and indistinct nuclei; the second variety shows larger round cells, delomorphous cells of Rollet,

also parietal cells, which in the fresh state show a finely granular contour, which becomes sharply outlined on addition of water or salt solution, they show coarsely grained contents and a distinct nucleus. The first form, the cubical or cylindrical chief or adelmorphous cells, are supposed to secrete the ferments pepsin and rennet; the second form, the larger round delomorphous cells or parietal cells, are supposed to secrete the HCL.

LECTURE II.

HISTOLOGY OF THE STOMACH AND INTESTINES.

THE PEPTIC GLANDS consist of a duct, a neck and a fundus; the latter is the deepest portion, and is often divided. These tubular glands have a distinct membrana propria separating them from the loose areolar connective tissue of the submucosa, in which they lie.

THREE KINDS OF CELLS OF THE PEPTIC GLANDS.

First, cylindrical cells of the gland duct and pit; lining $\frac{1}{4}$ to $\frac{1}{3}$ of the distance from the surface of the mucous membrane downward. A continuation of the cylindrical epithelium of the general internal surface of the gastric mucous membrane, these cells seem to secrete mucous only. Second, lightly colored, pyramidal or cuboidal cells with a granular protoplasm and spherical nucleus. These cells do not stain with aniline, and were termed adelmorphous cells by Rollet, — because they show no cell contours in the fresh state. Rosenheim states that they are almost clear and transparent during fasting, and become cloudy and granular during digestion. Haidenhayn designated them as the chief or central cells, and they were held by him to be the formers of the ferments, pepsinogen, and Rennet — Zymogen. These chief or central cells touch the lumen of the duct more extensively than the next following variety, the—Third, kind of peptic cells is known as the border, or partial cell, because they rest upon the membrana propria with much broader

bases than the chief or central cells, for this very reason they participate to a less degree in the limitation of the lumen of the duct; they are generally round or triangular, finely granular, and stain intensely with aniline, and were designated by Rollet as delomorphous cells; Haidenhayn supposes them to be the formers of hydrochloric acid. If we assume for the sake of locating these various cells; a division of the tubule into four sections, beginning at the portion nearest the submucosa, we shall have the (*a*) fundus of the gland tubule, then the (*b*) outer secretory portion, the (*c*) inner secretory portion and opening on the inner surface of the mucosa, the (*d*) alveolus;—Then one finds the border, parietal, delomorphous or aniline cells in the outer secreting portion most numerous, and becoming scarce in the fundus or end portion.

Haidenhayn asserted that there were no border cells in the fundus at all, but this has been denied by Stöhr, Kupffer and Boas. The number of border or acid cells depends upon the stage of digestion, as this function proceeds the border cells increase, and the chief, central, or ferment cells diminish in number, it would thus seem as if the border or acid cells, formed out of chief or ferment cells with the advance of digestion. In a fasting state the chief cells are largely in excess. Haidehayn's conclusions, that the chief or central cells are producers of the digestive ferments, and the border or aniline staining cells produce the hydrochloric acid, have been confirmed by a number of other observers (Grutzner, v Swiezicki, and recently Sehrwald).

Their method of experimentation was mainly the following; It is known that the glandular tubules of the pyloric region contain only chief or central cells, producing ferments only, and no acid, whilst the gland tubules of the fundus contain both central cells, and also border or acid cells. Now Haidenhayn succeeded in a number of dogs to dissect, and remove the pyloric portion of the stomach entirely, and to heal the organ into an external abdominal wound. In other dogs he removed the fundus entirely, leaving the pyloric portion intact, and succeeded in making this altered stomach without a fundus heal the external abdominal wound.

He therefore had two kinds of operated animals with stomachs opening on the abdomen. After this it was found that animals in which the pyloric region was excised furnished a juice that contained

both acid and pepsin, these were therefore produced by the glands of the fundus which contain both varieties of secretory cells. In the animals, however, that had been deprived of the fundus by excision, and the only secretory surface that was left being the pyloric region, it was found that an alkaline juice was secreted containing only ferments; that this juice did contain pepsin was proven by its power of digesting fibrin when hydrochloric acid was added to it.

Now as the gland tubules of the pylorus contain only chief or central cells, that do not stain with aniline, the conclusion is justifiable, that these chief cells secrete only ferments and that therefore the border or aniline staining cells must secrete the hydrochloric acid.

It has been found that the border or acid cells, called also the oxyntic cells,— are in communication with the central canal of the gland tubule by a tiny little canaliculus, an extension from the central lumen of the gland, to, or into the oxyntic or acid cells. These canaliculi were brought out by Golgi with his silver stain.

THE SMALL INTESTINE.

The small intestine commences at the pylorus, and, after many convolutions, terminates in the large intestine. It measures on an average, about 22 feet in length in an adult, and becomes gradually narrower from its upper to its lower end. Its convolutions occupy the middle and lower parts of the abdomen, and also frequently descend into the pelvis.

The small intestine is divided into three portions, which have received different names. The first ten to twelve inches immediately succeeding the stomach, and comprising the widest and most fixed part of the tube, is called the duodenum. This part is further distinguished by its close relation to the head of the pancreas, and by the absence of a mesentery. The remainder, which is arbitrarily divided into an upper two-fifths called the jejunum, and a lower three-fifths called the ileum, is very convoluted and movable, being connected with the posterior abdominal wall by a long and extensive fold of peritoneum called the mesentery, and by numerous blood vessels and nerves. Although there is no distinct line of demarcation between the jejunum and the ileum, yet the portion of the small intestine included under these two names gradually undergoes certain changes in structure and appearance from above downwards,

so that the upper end of the jejunum can readily be distinguished from the lower part of the ileum.

STRUCTURE OF THE SMALL INTESTINE.

The small intestine, like the stomach, is composed of four coats, viz., the serous or peritoneal, muscular, areolar, and mucous.

The external or serous coat almost entirely surrounds the intestinal tube in the whole extent of jejunum and ileum leaving only a narrow interval behind, where it passes off and becomes continuous with the two layers of the mesentery. The line at which this takes place is named the attached or mesenteric border of the intestine. The duodenum, on the other hand, is but partially covered by the peritoneum. The muscular coat consists of two layers of fibers; an outer longitudinal, and an inner or circular set. The longitudinal fibers constitute an entire but comparatively thin layer, and are most obvious along the free border of the intestine. The circular layer is thicker and more distinct.

The muscular tunic becomes gradually thinner towards the lower part of the small intestine. It is pale in color, and is composed of plain muscular tissue, the cells of which are of considerable length.

The progressive contraction of these fibers, commencing at any part of the intestine, and advancing in a downward direction, produces the peculiar vermicular or peristaltic movement by which the contents are forced onwards through the canal. In the narrowing of the tube the circular fibers are mainly concerned, the longitudinal fibres tending to produce dilatation (Exner); and those found along the free border of the intestine may have the effect of straightening or unfolding its successive convolutions. There is a gangliated plexus of nerve-fibres and a network of lymphatic vessels between the two muscular layers.

The submucous coat of the small intestine is a layer of areolar tissue of a loose texture, which is connected more firmly with the mucous than with the muscular coat. Within it the blood-vessels ramify before passing to the mucous membrane, and there is a gangliated plexus of nerve-fibers and a network of large lymphatic vessels in it.

The internal coat or mucous membrane is characterised by the finely flocculent or shaggy appearance of its inner surface resembling

the pile upon velvet. This appearance is due to the surface being thickly covered with minute processes named villi. It is one of the most vascular membranes in the body, and is naturally of a reddish color in the upper part of the small intestine, but is paler, and at the same time thinner, towards the lower end. It is lined with columnar epithelium throughout its whole extent, and next to the submucous coat is bounded by a layer of plain muscular tissue (*muscularis mucosa*); between this and the epithelium the substance of the membrane, apart from the tubular glands which will be afterwards described, consists mainly of retiform tissue which supports the blood-vessel, nerves, lymphatics and lacteals, and encloses in its meshes numerous lymph-corpuscles.

VALVULÆ CONNIVENTES.

The mucous membrane, in addition to small effaceable folds or rugae, possesses also permanent folds, which cannot be obliterated, even when the tube is forcibly distended. These permanent folds are the *valvulæ conniventes* or valves of Kerkring. They are crescentic projections of the mucous membrane, placed transversely to the axis of the bowel and following one another closely. The majority of the folds do not extend more than one-half or two-thirds around the interior of the tube, but it has been shown by Brooks and Kazzander that some form complete circles, and others spirals. The spiral forms may occur singly or in groups of two or three. They generally extend a little more than once around the bowel, but in rare cases may go round two or three times. At their highest point they project inwards for about a third of an inch. Some of the *valvulæ conniventes* are bifurcated at one or both ends, and others terminate abruptly. Each consists of a fold of mucous membrane, that is, of two layers placed back to back, and united together by submucous areolar tissue. They contain no part of the circular or longitudinal muscular coats. Being extensions of the mucous membrane they serve to increase the absorbent surface to which the food is exposed.

The *valvulæ conniventes* are not uniformly distributed over the various parts of the small intestine. There are none quite at the commencement of the duodenum; a short distance from the pylorus they begin to appear; beyond the point at which the bile and pan-

creatic juice are poured into the duodenum they are very large, regularly crescentic in form, and placed so near to each other that the intervals between them are not greater than the breadth of the valves; they continue thus through the rest of the duodenum and along the upper half of the jejunum; below that point they begin to get smaller and farther apart, and finally, towards the middle or lower end of the ileum, having gradually become more irregular and indistinct, sometimes even acquiring a very oblique direction, they altogether disappear.

The villi, peculiar to the small intestine, and giving to its internal surface the velvety appearance already spoken of, are small processes of the mucous membrane, which are closely set on every part of the inner surface over the *valvulæ conniventes*, as well as between them. Their length varies from 0.5 mm. to 0.7 mm. or sometimes more.

They are largest and most numerous in the duodenum and jejunum, and becomes gradually smaller, and fewer in number in the ileum. According to Rauber, they are short and leaf-shaped in the duodenum, and as the gut is followed downwards they become gradually longer and thinner, so that they are tongue-shaped in the jejunum, and filiform in the ileum. Occasionally two or three are connected together at their bases. In the upper part of the small intestine there are from 10 to 18 villi in a square millimeter, and in the ileum from 8 to 14 in the same space. This would give about four millions altogether (Krause.)

A villus consists of a prolongation of the proper mucous membrane. It is covered by columnar epithelium, and encloses a network of blood-vessels, one or more lymphatic vessels (*lacteals*), and a few longitudinal plain muscular fiber-cells, these being all supported and held together by retiform lymphoid tissue.

Under the epithelium is a basement membrane composed of flattened cells, which on the one hand are connected with the branched cells of the retiform tissue and on the other hand send processes between the epithelial cells. Nervous fibrils penetrate into the villi from the plexus of Meissner and form arborizations throughout their whole substance.

Each villus receives as a rule one small arterial twig, which runs from the submucous coat, through the *muscularis mucosae* to the

base of the villus and then up the centre to near the middle line of the villus where it begins to break up into a number of capillaries.

These form near the surface, beneath the epithelium and limiting membrane, a fine capillary network, from which the blood is returned for the most part by one or two venules, which in man commence near the tip of the villus and pass down to its base to join the venous plexus of the mucous membrane, whence the blood is conveyed to the large veins of the submucosa.

The lacteal lies in the centre of the villus and in the smaller villi is usually a single vessel with a closed and somewhat expanded extremity and of considerably larger diameter than the capillaries of the blood vessels around. In the human subject there are never more than two intercommunicating lacteals in a single villus.

The lacteals in the villi are bounded by a delicate layer of flattened epithelial cells; these are connected with the branched cells of the tissue of the villus, and these again with the flattened cells forming the basement membrane; from the latter, prolongations extend between the epithelium cells toward the surface. Brücke first discovered the muscular tissue within the villus, consisting of unstriated plain fibre cells, disposed longitudinally around the lacteal. In animals a very evident retraction of the villus is noticeable when these muscular fibers which are prolongations from the muscularis musosae are stimulated.

The fiber cells at the sides and towards the end of the villus pass from the lacteal to be attached to the basement membrane in a bifurcating manner.

Columnar epithelial cells cover not only the villi but also the rest of the surface of the small intestine and extend into the tubular glands. There is never any continuity between the extremity that is attached to the basement membrane and the branched corpuscles of the retiform tissue of the villus. This epithelium separates easily from the subjacent tissue. Between the cells composing it are a variable number of leucocytes most numerous in the lower part of the intestines near the lymphoid follicles. Occasionally they are seen to be free in small lymph spaces between the columnar epithelial cells and showing indications of Karyokinesis. Hardy declares that immediately below the columnar epithelium of the villi, there is frequently a well marked layer of cells that take up the eosin stain

readily. Hence, he calls them eosinophilic.

LECTURE III.

HISTOLOGY OF INTESTINAL MUCOSA.

AMONG the ordinary epithelial cells of the villus are others, the outer half of which is filled with mucigen, and at times such beaker or cup shaped empty cells are observed from which this has been discharged as mucous, and the free end is ruptured; these are sometimes called the goblet cells. The number of cells containing mucous varies much in different animals and under different conditions in the same animal. There are comparatively few mucous cells in the glands of the small intestine.

The epithelial cells are as far as can be ascertained the principal agents in promoting the absorption of food materials from the interior of the gut, and the seat of the retrograde processes of metabolism which the products of digestion undergo during absorption. Peptone when injected into the blood of an animal whose gastric juice has formed, it acts as a poison. It is due to these epithelial cells of the intestine that peptone is so modified by them during absorption that it becomes of use to the organism.

Most food particles can not be traced in microscopic specimens, but fatty or oily substances, from their property of becoming stained with osmic acid can be to some extent followed out. The examination of such specimens taken during digestion of a meal containing fat, shows the epithelial cells turbid with oil droplets in their interior, and in some animals at a subsequent stage, amoeboid cells appear within the tissue of the villus pervaded with similar but finer fatty particles and eventually the central lacteal becomes filled with these. It is probable that these amoeboid lymph corpuscles appearing so abundantly within the villus and among the epithelial cells on its surface, play an important part in the transference of such particles from the epithelial cells in the lacteal, for at certain stages

of fat absorption. they contain abundant fatty particles. The large amount of lymphoid tissue in the lower part of the small intestine seems to be related to a greater power of absorption in that part.

In the transference of carbon particles in the lungs, from the interior of the alveoli into the lymphatics, which at least in part is due to the action of amoeboid cells, we have an analogous process.

GLANDS.

Two kinds of true secreting glands are found in the intestine these are; (1) Glands or crypts of Lieberkühn and the (2) glands of Brunner. In addition to these, there are found also two varieties of intestinal lymph follicles the (1) Solitary and (2) the Agminate glands, the latter often designated as Peyer's patches.

Although the solitary and agminated lymph follicles have no ducts opening upon the inner intestinal surfaces, like Brunner's and Lieberkühn's glands, they are nevertheless spoken of as glands.

The follicles crypts or glands of Lieberkühn are tubular pits lined by columnar epithelium, occurring between the villi and on the valvulæ conniventes. Here and there in these crypts, goblet cells occur in the epithelium. They are present throughout the large and small intestine, and extend through the entire depth of the mucosae, their ends approaching the muscularis mucosae.

The duodenum possesses an additional layer of true secreting structures in the glands of Brunner. They would appear to represent the direct continuations and higher specializations of the pyloric glands. In passing from the stomach into the intestines, these tubules undergo repeated division, at the same time sinking deeper into the mucosa, finally reaching below this layer to take up a position within the submucosa of the duodenum, underneath the overlying layer of the crypts of Lieberkühn which are contained in the mucosa proper. Brunner's glands belong to the racemose type and under the microscope they consist of a number of tubular alveoli connected by terminal ramifications of the duct which penetrates the muscularis mucosae, and opens either between the mouths of the Lieberkühn crypts or sometimes into their bases.

The solitary glands are isolated lymph follicles scattered through the entire intestine, most abundant in lower ileum. Situated in the mucosa, at times in the submucosa; the lymphoid tissue in them is

denser toward the periphery, but is everywhere so closely packed that the supporting reticulum of connective tissue is masked.

The agminated glands, or Peyer's patches are large oval groups of closely aggregated groups of lymph follicles held together by diffuse adenoid tissue, limited to lower $\frac{2}{3}$ of the small intestine. Development of these is most perfect in the ileum, appearing first within the mucosa, they later encroach upon the submucous tissue.

Where the summits of these follicles impinge against the inner layer of the mucosa, the position of the agminated glands are indicated by an elevation corresponding to them on the mucous surface. In that case the villi are frequently pushed aside..

THE BLOOD-VESSELS OF THE INTESTINES.

The vessels follow the general arrangement of those in the stomach; the larger ones piercing the serous and muscular coat, giving off slender twigs to supply these tunics, and when they enter the submucosa, the vessels form a wide, meshed, network. Many branches then pass through the muscularis mucosae to be distributed to the deeper, as well as the superficial part of the mucosa. Around the tubular glands a network is formed by narrow capillaries, and just beneath the epithelium the capillaries become wider and encircle the mouths of the follicles. From this superficial capillary network the veins arise, and passing down between the follicles, join the deeper venous plexus, this in turn communicating with the larger veins of the submucosa.

The villi have special additional arteries running to their bases, expanding into capillaries that extend beneath the epithelium and around the central lacteal as far as the ends of the villi. These capillaries terminate in venous stems which descend almost perpendicularly into the mucosa in their course receiving the superficial capillaries encircling the gland ducts. Brunner's glands, and the solitary and agminated follicles are supplied from the submucosa by vessels terminating in capillary networks distributed to the acini of the glands and interior of the lymph follicles.

The blood-vessels of the intestines taken as a whole, constitute a mighty vascular territory which is capable of taking up $\frac{1}{3}$ of the total amount of blood of the body.

The arteries are all branches of the superior and inferior mesen-

teric arteries which run along and approach the gut in the mesentery. The intestinal veins form the principal portion of the portal artery system.

LYMPH-VESSELS.

The beginning of the lymph-vessels can be traced to the lacteals within the villi, where they begin as tiny little blind pouches at the apex of the villus. In some broad villi there are two or three such lymph-vessels that anastomose with one another. From here they run down in the septa between the glands in the lymph-vessel mesh-work over the muscularis mucosae. This again anastomoses with an outer lymph-vessel network in the submucosae. Here already, the lymphatics begin to be provided with valves.

The nerves of the intestine, like those of the stomach originate chiefly from the mesenteric plexus which is formed by branches from the coeliac plexus, the semilunar ganglion and vagus nerve. These nerve branches consist of medullated and nonmedullated fibers, that begin to form an abundant network under the peritoneum of the intestine, then penetrate the longitudinal muscular stratum and between this and the circular layer form a peculiar plexus with numerous microscopic ganglia; they are called the plexus of Auerbach.

In the submucosa a similar network of fibers and ganglia has been termed Meissner's plexus. From Meissner's plexus very fine fibers are spun about the Lieberkühn crypts, villus and limiting membrane.

RELATIONS OF THE DUODENUM.

This part of the gut in the adult is horse-shoe shaped, generally presenting well marked angles which divide it into four parts having four distinct directions, these are the (1) horizontal or superior part running backwards from the pylorus, to the right, in contact with the quadrate lobe of the liver, to the underside of the neck of the gall bladder where it curves sharply downwards to join the second part. This first or horizontal part is about 2 inches long when the stomach is empty. (2) The second or descending portion is about three inches long and commences just below the neck of the gall bladder opposite the right side of the first lumbar vertebra and passes down to the level of the third or fourth lumbar vertebra

where it turns sharply inwards to join the third part. (3) The third or transverse portion is between two to three in. long; beginning at the right of the third or fourth lumbar vertebra, it crosses over to the left side with a slight upward inclination and ends to the left of the aorta by curving upwards to join the terminal (4) fourth or ascending portion which is about two inches long; it passes upward to the left side of the aorta as high as the upper border of the second lumbar vertebra; here it turns abruptly forwards to join the jejunum,



THE DUODENUM.

forming the duodenojejunal flexure.

Thus the end of the duodenum is brought to the same level as the beginning. It has been compared to a trap its ends being always higher than its middle, which is thus fitted to retain the fluid poured into it from the liver, pancreas and its own glands, besides that which it receives from the stomach.

JEJUNUM AND ILEUM.

The upper two-fifths of the remaining intestine immediately following the duodenum are called the jejunum, the lower three-fifths of the remainder, the ileum. Both are attached to the posterior abdominal wall by an extensive fold of peritoneum and the mesentery.

The jejunum lies above and to the left of the ileum, but the coils are so irregular, that the position of any individual loop affords but little clue to the part of the intestine to which it belongs.

The large intestine consists of the coecum, the colon and the rectum. The colon is subdivided according to the directions it takes into four parts which are the (1) ascending, (2) transverse, (3) descending and (4) sigmoid colon or flexure.

The end of the ileum which rises out of the pelvis to the right ileac fossa, is not inserted into the beginning of the large intestine, but above the beginning and at the side of it. The part of the large intestine below this insertion is a blind pouch, the coecum. From the inner and back part of the coecum a little below the ileo colic opening, a narrow, round, worm like process about two or three inches long is given off, the vermiform appendix.

The coecum continues upward into the ascending colon which rises up in front of the right kidney to the edge of the liver, then this same large intestine passes beneath the greater curvature of the stomach, horizontally across to the left side as the transverse colon, here at the lower border of the spleen it turns downward as the descending colon.

This large gut describes two right angled curves, the right and left colonic flexures fixed by the hepato colic and gastro colic ligaments respectively. The descending colon continues into the sigmoid colon or flexure which connects it with the rectum. The rectum following the curves of the sacro iliac symphysis and of the hollow of the sacrum itself has two curves;—an upper larger curve, concave anteriorly and a lower smaller curve, convex anteriorly.

Only coecum, transverse colon and sigmoid have a complete peritoneal covering, the rest of the large gut is only covered anteriorly. From the third sacral vertebra on, the rectum has no peritoneum. Those parts having no complete peritoneum, therefore have

no mesentery and are not very movable. The longitudinal fibers are contracted or narrowed down to 3 parallel bands. (Fasciae Taeniae or lig. coli.) One of these bands runs along the attachment of the gastro colic ligament on the transverse colon, [fascia omentalis] the second along the mesenteric border and the third is free.

Running down into the rectum these bands become so broad that they occupy the entire periphery of the tube. These longitudinal bands are much shorter than the other layers of the intestinal wall, which arrangement results in the characteristic sacculation of the large intestine. In the lower part of the rectum the circular muscular layer becomes thickened to form the internal anal sphincter of involuntary fibers.

The external sphincter is composed of striated voluntary muscle fibers. The histology of the large, differs from that of the small intestine by the absence of the villi and larger size of the crypts and follicles. Several longitudinal elevations over the anus are called the columns of Morgagni; from this point downward the cylindrical epithelium ceases and flat pavement epithelium takes its place.

LECTURE IV.

PHYSIOLOGY OF DIGESTION.

THE SIMPLE chemical elements of the various food substances namely C. H. N. S. and P. are not assimilable as such, because the human body is not capable of constructing higher compounds from them synthetically. It is compelled to take in these compounds in form of proteid or albuminous substances, carbohydrates or starches and fats together with such inorganic bodies as water and salts.

Even these foodstuffs which are essential for the maintenance and developement of the organism are not ingested as such but are contained together with innutritious materials in the various articles of diet which we derive from the animal and vegetable kingdom.

The innutritious admixtures of the food substances are not harmful, but are important as stimulants to the intestinal mucosa and to evacuation of feces. Among these innutritious substances are classed the connective tissue, the cartilages and tendons of meat and the cellulose of plants.

Water plays a most important role in the economy of the body, for it goes to make up 60 per cent of the total organism. We lose about $2\frac{1}{2}$ litres of water in 24 hours, through insensible perspiration, secretion and by defecation. About 300-400 grammes of water are formed by oxidation of food substances in 24 hours; so we have a deficit of 1500-1600 grammes, which must be supplied by the daily consumption of a corresponding amount of water; this occurs principally by the use of drinking water after we have taken in part of it by our foods or in shape of beverages. (Soups, milk, fruits, vegetables, potatoes, beer, wine, coffee, tea, etc.)

Of mineral substances we must supply the daily loss of sodium chloride and other salts, particularly compounds of iron; these are normally introduced in sufficient quantities in food and drink.

The chief constituents of food, Albuminous bodies, fats and carbohydrates, are of organic nature. The proteids or albuminous bodies and the fats are derived partly from the animal and partly from the vegetable kingdom. The carbohydrates are almost exclusively derived from the vegetable kingdom. The former serve the building up of the organism, and the continuance of life processes. The latter are producers of heat by their oxidation, which finally reaches HCO_3 and H_2O and are the prevailing sources of work.

In addition to these a number of other substances occur in the food that are oxydized and might serve as sources of energy, these are the nitrogen free vegetable acids, the amido acids and alcohol for instance; quantitatively however they are not important.

Other organic bodies that are contained in food materials as normal constituents, are Kreatin in meat, Glucosides, Alkaloids and ethereal oils in vegetables and spices, pass through the body without being oxydized or assimilated; they are not food substances, as they do not enter the metabolism of the body nor do they develop energy by chemical transformation. However a number of these are of importance in nutrition as they render the food more palatable and stimulate the secretions and the motility of the diges-

tive tract.

In another place I said that the elements S.P.Cl.K.Na.Ca.Fe.Mg., are not food materials, but it must not be understood that they are entirely useless. They have some significance in the construction of tissue, although the organism can derive no energy from them, as they are always taken, in a highly oxydized state and leave in the same condition. Nevertheless the body will come to grief, if any one of these elements is excluded from the food.

A certain minimum of these elements—the amount has not yet been ascertained—is absolutely necessary. Outside of the substances named above the food contains a number of materials, that are not at all absorbable or digestable and leave the digestive tract in an unchanged form; this is the slag and dross of the food and is taken into the body with vegetables.

The normal, adult human organism daily loses by its metabolism, 120 grms. of Albuminous or proteid bodies, 80 g. fat, 400 g. carbohydrates, 25 g. salts and 2½ litres of water. Accordingly a corresponding amount of foodstuffs must be introduced in the diet. The articles of food contain these nutritious substances in a variety of proportions. The rational combination of these substances is one of the objects of dietetics. Gilman Thompson in his new book on Dietetics, divides foods into six groups as follows; i. Water, ii. Salts, iii. Proteids chiefly albuminous and allied gelatine, iv. Starches, v. Sugar, vi. Fats and oils.

It still remains extremely difficult in the case of all foods, to trace their final uses in the body and determine with any accuracy, what proportions of each furnish respectively, energy, repair of tissue and heat, for there are no more complex chemical processes known than those of metabolism. [Gilman Thompson] Foods have three kinds of values: (1) Nutrient, (2) heat producing, (3) force producing values.

The calculation of these different values, for each kind of food has been much simplified by the introduction of the conception of calories into the doctrines of nutrition. Formerly observers and investigators said, "A healthy man needs so many grms. proteid, so many grms. carbohydrates, so many grms. fat, etc. It was inconvenient to reckon with three magnitudes and to bring them into correct relation with the requisites of the individual organism.

Nowadays we compute the values of foodstuffs according to the physiological (kinetic) energy liberated in their oxidation. Germans call this degree of energy which is always expressed in terms of heat the "Brennwerth," that means the value of food when it is burned in the process of metabolism, for this is nothing but a slow combustion. Now the unit for measurment of this heat energy of food is called a calorie. This capacity for heat production of foods is determined from the amount of heat which is liberated when any particular food substance is transformed from its original composition when it entered the body,—by oxidation,—into those chemical combinations in which it leaves the organism. The unit for measurment is the calorie which signifies the amount of heat which is necessary to raise 1 kilogram of water, 1° centigrade.

Now	1 gram of Albumen	furnishes	— 4.1	Calories.
	1 " Carbohydrate	"	4.1	"
	1 " Fat	"	9.3	"
	1 " Alcohol	"	7.	"

Instead of saying a man requires 100 grms. Albumen, 100 grms. Fat and 400 grms. Carbohydrate, one now expresses this in calories thus;—A man requires

100 gr. Albumen	x	4.1	——	410	Calories.
100 gr. Fats	x	9.3	——	930	"
400 gr. Carbohydrate	x	4.1	——	1640	"
Total				2980	"

For every kilogram of body weight an adult requires,				
when at rest, —— a food supply of			30—34	Calories.
During light occupation	"	"	34—40	"
" medium "	"	"	40—45	"
" hard work	"	"	45—60	"

In very adipose and obese person the requirements for food is less than the quantities stated by $\frac{1}{4}$ to $\frac{1}{3}$. If the above calculations of requisite number of calories per kilogram weight of any person is correct, and the supply maintained accordingly, the individual will maintain his weight. If the supply of calories is greater, he will gain weight, if the supply is less he will loose weight.

In a condensed statement of facts like the present, it will be expedient to pass over the physiology of hunger, appetite and thirst,

and proceed at once to digestive actions.

Digestion really begins in the mouth, where the food is chewed into small bits and is mixed with saliva, which mechanically facilitates mastication and deglutition. Chemical transformation also begins here, for the diastasic ferment of saliva the Ptyalin transforms a small portion of the starchy foods into sugar and maltose.

Ptyalin can produce this transformation of starchy foods only in an alkaline medium, accordingly the action ceases in the stomach but not immediately however, as the conversion of starches into sugar goes on until the degree of acidity reaches 1 to the thousand, (1 pro mille.) As the ptyalin ferment becomes inactive in this acidity, the question arises, whether its activity is permanently destroyed by 1 pro mille acid or only temporarily and whether it can resume its inverting power when the acid is neutralized. Boas who attempted a solution of this, came to the conclusion that subsequent alkalization or diminution of the acid, causes the ptyalin to act again, so that in later stages of stomach digestion when the acid production ceases, the conversion of starch into grape sugar may be resumed.

The existence of appetite is largely dependant upon the intactness of the salivary glands. In order to understand the various stages of starch conversion, it is essential for you to study the digestion of starch by ptyalin in the clinical laboratory. There are recognized four stages of starch conversion, each distinct from the other until dextrose is reached.

1. (a) These are common starch representing a glue like mucilagenous jelly, not a clear solution giving a dark blue color with iodine and iodide of potassium solution. The next stage shows the first action of ptyalin.

1. (b) AMIDULIN OR AMYLODEXTRIN: This still gives a distinctly blue color though not so deep as No. 1a with Lugols solution—but Amylodextrin is a soluble starch and represents a real solution.

2. (a) ERYTHRO DEXTRIN:—Gradually as the inversion progresses the color produced by the iodine solution becomes violet blue, violet, red violet, red or mahogany brown; this modification is called erythro dextrin. (b) ACHROODEXTRIN:—With continued action of the ptyalin, a substance is reached which gives no color with iodine; this is called achroodextrin. Amidulin is precipitated by

Tables of Dietetics.

Approximate Analyses of a Man (Moss).

(Height, 5 feet 8 inches; weight 148 pounds.

Oxygen	—	—	—	—	92.4 pounds.
Hydrogen	—	—	—	—	14.6 “
Carbon	—	—	—	—	31.6 “
Nitrogen	—	—	—	—	4.6 “
Phosphorus	—	—	—	—	1.4 “
Calcium	—	—	—	—	2.8 “
Sulphur	—	—	—	—	0.24 “
Chlorine	—	—	—	—	0.12 “
Sodium	—	—	—	—	0.12 “
Iron	—	—	—	—	0.02 “
Potassium	—	—	—	—	0.34 “
Magnesium	—	—	—	—	0.04 “
Silica	—	—	—	—	?
Fluorine	—	—	—	—	0.02 “
Total					148.00 pounds.

Landois and Sterling give the following table, which differs somewhat from the other tables in the relative proportion of fats and starches. An adult doing a moderate amount of work takes in as food per diem—

	C.	H.	N.	O.
120 grammes albumin, containing	64.18	8.60	18.88	28.34
90 grammes fats, containing	70.20	10.26	9.54
330 grammes starches, containing	146.82	20.33	162.85
	281.20	39.19	18.88	200.73

Add 744.11 grammes O. from the air by respiration.

" 2,818.00 " H₂O.

" 32.00 " inorganic compounds [salts].

The whole is equal to three kilogrammes and a half [seven pounds], *i. e.*, about a twentieth of the body weight, so that about six percent of the water, about six percent of the fat, about one percent of the albumin, and about 0.4 percent of the salts of the body are daily transformed within the organisms.

An adult doing a moderate amount of work gives off in grammes:

	Water.	C.	H.	N.	O.
By respiration	330	248.8	?	651.15
By perspiration	660	2.6	7.2
By urine	1,700	9.8	3.3	15.8	11.1
By faeces	128	20.0	3.0	3.0	12.0
	2,818	281.2	6.3	18.8	681.45

Standards for Daily Dietaries.

(Compiled by Atwater.)

Weights of nutrients and calories of energy [heat units] in nutrients required in food per day.

	NUTRIENTS.				Potential energy.
	Protein.	Fats.	Carbohydrates.	Total.	
	<i>Grms.</i>	<i>Grms.</i>	<i>Grms.</i>	<i>Grms.</i>	<i>Calories.</i>
Children to a year and a half	28 (20-36)	37 (30-45)	75 (60-90)	140	767
Children of two to six years—	55 (36-70)	40 (35-48)	40 (100-250)	295	1,418
Children of six to fifteen years	75 (70-80)	43 (37-50)	325 (250-400)	443	2,041
Aged women	80	50	260	390	1,859
Aged man	100	68	350	518	2,477
Women at moderate work <i>Voit</i>	92	44	490	536	2,426
Man at moderate work, <i>Voit</i>	118	56	500	674	3,055
Man at hard work, <i>Voit</i>	145	100	450	695	3,370
Man at moderate exercise, <i>Playfair</i>	119	51	531	701	3,139
Active labor, <i>Playfair</i>	156	71	568	795	3,629
Hard labor, <i>Playfair</i>	185	71	568	824	3,748
Women with light exercise, <i>Atwater</i>	80	80	300	460	2,300
Man with light ex'rc. <i>Atwater</i>	100	100	360	460	2,820
Man at moderate work, "	125	125	450	700	3,520
Man at hard work, "	150	150	500	800	4,060
Man at moderate " <i>Moleschott</i>	130	40	550	720	3,160
Man at moderate work, <i>Wolff</i> .	120	35	540	695	3,032

Table of analyses made by Dujardin-Beaumetz showing the proportion of nitrogen present and also the combustible carbon and hydrogen.

	Nitrogen.	C + H Combustibles cal- culated as carbon.
Beef (uncooked).....	3.00	11.00
Roast beef.....	3.53	17.76
Calf's liver.....	3.09	15.68
<i>Foie gras</i>	2.12	65.58
Sheep's Kidneys.....	2.66	12.13
Skate.....	3.83	12.25
Cod, salted.....	5.02	16.00
Herring, salted.....	3.11	23.00
Herring, fresh.....	1.83	21.00
Whiting.....	2.41	9.00
Mackerel.....	3.74	19.26
Sole.....	1.91	12.25
Salmon.....	2.00	16.00
Carp.....	3.49	12.10
Oysters.....	2.13	7.18
Lobster (uncooked).....	2.93	10.96
Eggs.....	1.90	13.50
Milk [cow's].....	0.66	8.00
Cheese [Brie].....	2.93	35.00
Cheese [Gruyère].....	5.00	38.00
Cheese [Roquefort].....	4.21	44.44
Chocolate.....	1.52	58.00
Wheat (hard southern, variable average).....	3.00	41.00
Wheat (soft southern, variable average).....	1.81	39.00
Flour, white [Paris].....	1.64	38.50
Rye flour.....	1.75	41.00
Winter barley.....	1.90	40.00
Maize.....	1.70	44.00
Buckwheat.....	2.20	42.50
Rice.....	1.80	41.00
Oatmeal.....	1.95	44.00
Bread, white (Paris, thirty per cent water).....	1.08	29.50
Bread, brown (soldiers' rations formerly).....	1.07	28.00
Bread, brown (soldiers' rations at present).....	1.20	30.00
Bread from flour of hard wheat.....	2.20	31.00
Potatoes.....	0.33	11.00
Beans.....	4.50	42.00
Haricots [dry].....	3.92	43.00
Lentils [dry].....	3.87	43.00
Peas [dry].....	3.66	44.00
Carrots.....	0.31	5.50
Mushrooms.....	0.60	4.52
Figs [fresh].....	0.41	15.50
Figs [dry].....	0.92	34.00
Plums.....	0.75	28.00
Coffee (infusion of 100 grammes).....	1.10	9.00
Tea (infusion of 100 grammes).....	1.00	10.50
Bacon.....	1.29	71.14
Butter [fresh].....	0.64	83.00
Olive oil.....	98.00
Beer, strong.....	0.05	4.50
Wine.....	0.15	4.00

The Relative Value of Foods [Scammell.]

(The figures represent percentages.)

ARTICLES.	As material for the muscles.	As heat givers.	As food for the brain and nervous system.	Water.	Waste.
Wheat	14.6	66.4	1.6	14.0	3.4
Barley	12.8	52.1	4.2	14.0	16.9
Oats	17.0	50.8	3.0	13.6	16.9
Northern corn	12.3	67.5	1.1	14.0	5.1
Southern corn	34.6	39.2	4.1	14.0	8.1
Buckwheat	8.6	53.0	1.8	14.2	22.4
Rye	6.5	75.2	0.5	13.5	4.3
Beans	24.0	40.0	3.5	14.8	17.7
Peas	23.4	41.0	2.5	14.1	19.0
Lentils	26.0	39.0	1.5	14.0	19.5
Rice	5.1	82.0	0.5	9.0	3.4
Potatoes	1.4	15.8	0.9	74.3	7.1
Sweet Potatoes	1.5	21.8	2.9	67.5	6.3
Parsnips	2.1	14.5	1.0	79.4	3.0
Turnips	1.2	4.0	0.5	90.4	3.9
Carrots	1.1	12.2	1.0	82.5	3.2
Cabbage	1.2	6.2	0.8	91.3	0.5
Cauliflower	3.6	4.6	1.0	90.0	0.8
Cucumbers	0.1	1.7	0.5	97.1	0.6
Milk of cow	5.0	8.0	1.0	86.0	—
Milk, human	3.0	7.0	0.5	89.5	—
Veal	17.7	14.3	2.3	65.7	—
Beef	19.0	14.0	2.0	65.0	—
Lamb	19.6	14.3	2.2	63.9	—
Mutton	21.0	14.0	2.0	63.0	—
Pork	17.5	16.0	2.2	64.3	—
Chicken	21.6	1.9	2.8	73.7	—
Codfish	16.5	1.0	2.5	80.0	—
Trout	16.9	0.8	4.3	78.0	—
Smelt	17.0	very little	5 or 6	75.0	—
Salmon	20.0	some fat	6 or 7	74.0	—
Eels	17.0	"	3 or 4	75.0	—
Herring	18.0	"	4 or 5	75.0	—
Halibut	18.0	"	3 or 4	74.0	—
Oysters	12.6	—	0.2	87.2	—
Clam	12.0	very little	2 or 3	—	—
Lobster	14.0	"	5 or 6	79.9	—
Eggs (white of)	13.0	—	2.8	84.2	—
Eggs (yolk of)	—	29.8	2.0	51.3	—
Butter	—	100.0	—	—	—
Artichoke	1.9	19.0	1.8	76.6	0.7
Asparagus	0.6	5.4	0.4	93.6	—
Bacon	8.4	62.5	0.5	28.6	—
Carp	18.0	0.8	2.9	78.3	—
Cheese	30.8	28.0	4.7	36.5	—
Cherries	0.6	21.0	1.0	76.3	1.1
Chocolate	8.8	88.0	1.8	—	1.4
Cream	3.5	4.5	—	92.0	—

ARTICLES.	As material for the muscles.	As heat givers.	As food for the brain and nervous system	Water.	Waste.
Currants.....	0.9	6.8	0.3	81.3	10.7
Dates (fresh).....	—	73.7	—	24.0	2.3
Figs.....	5.0	57.9	3.4	18.7	15.0
Ham.....	35.0	32.0	4.4	28.6	—
Horseradish.....	0.1	4.8	1.0	78.2	16.0
Kidney.....	21.2	0.9	1.4	76.5	—
Lard.....	—	100.0	—	—	—
Liver.....	26.3	3.9	1.2	68.6	—
Onions.....	0.5	5.2	0.5	93.8	—
Pearl barley.....	4.7	78.0	0.2	9.5	7.6
Pears.....	0.1	9.6	—	86.4	3.9
Pigeon.....	23.0	1.9	2.7	72.4	—
Prunes.....	3.9	78.6	4.5	13.0	—
Radishes.....	1.2	7.4	1.0	89.1	1.3
Suet.....	—	100.0	—	—	—
Venison.....	20.4	8.0	2.8	68.8	—
Vermicelli.....	47.5	38.0	1.7	12.8	—
Whey.....	—	4.6	0.7	94.7	—

Atkinson's Table of Digestibility of Nutrients of Food Materials.

IN THE FOOD MATERIALS BELOW	OF THE TOTAL AMOUNTS OF PROTEIN, FATS, AND CARBOHYDRATES THE FOLLOWING PERCENTAGES WERE DIGESTED.		
	Protein.	Fats.	Carbohydrates.
Meat and fish.....	Practic'ly all	79 to 92	—
Eggs.....	—	96	—
Milk.....	88 to 100	93 to 98	?
Butter.....	—	98	—
Oleomargarine.....	—	96	—
Wheat Bread.....	81 to 100	?	99
Corn [maize] meal.....	89	?	97
Rice.....	84	?	99
Peas.....	86	?	96
Potatoes.....	74	?	92
Beets.....	72	?	82

Percentages of Nutrition in various Articles of Food. (Moss.)

Raw Cucumbers.....	2	Raw beef.....	26
Raw melons.....	3	Raw Grapes.....	27
Boiled turnips.....	4½	Raw prunes.....	29
Milk.....	7	Boiled mutton.....	30
Cabbage.....	7½	Oatmeal porridge.....	75
Currants.....	10	Rye bread.....	79
Whipped Eggs.....	13	Boiled beans.....	87
Beets.....	14	Boiled rice.....	88
Apples.....	16	Barley bread.....	88
Peaches.....	20	Wheat bread.....	90
Boiled Codfish.....	21	Baked corn bread.....	91
Boiled venison.....	22	Boiled barley.....	92
Potatoes.....	22½	Butter.....	93
Fried veal.....	24	Boiled peas.....	93
Roast Poultry.....	26	Raw oil.....	96

It will be observed that the totals are somewhat less in this diet than those of the preceeding table, which is designed for a working man who is developing more calories.

TABLE OF ENERGY

estimated in Foot Tons instead of Calories. (YEO.)

Energy developed by one ounce of the following foods when oxidised in the body.

FOOD STUFF.	With usual percentages of water	One ounce water-free.
	<i>Foot tons.</i>	<i>Foot tons.</i>
Beef [best quality], uncooked	48.5	199
Meat [served to soldiers], uncooked	57.8	243
Beef [fattened], uncooked	96.0	280
Meat, cooked	102.6	240
Corned beef [Chicago]	124.0	217
Salt beef	52.0	138
Salt pork	71.6	166
Fat pork	202.0	336
Dried bacon	292.3	346
Smoked ham	179.6	267
Whitefish	44.3	209
Poultry	50.7	204
Bread	87.5	147
Wheat flour	123.6	146
Biscuit	173.3	189
Rice	126.5	141
Oatmeal	130.0	154
Maize	132.0	160
Macaroni	122.7	146
Millet	125.9	149
Arrowroot	116.4	138
Peas [dried]	118.9	151
Potatoes	33.0	141
Carrots	14.3	137
Cabbage	13.0	158
Butter	344.5	367
Eggs	67.3	265
Cheese	149.9	245
Milk [cow's], new	26.9	225
Cream	109.2	365
Skimmed milk	20.4	181
Sugar	126.4	128
Penmican	270.1	293
Ale [Bass's bottled]	30.0	260
Stout [Guinness]	41.5	360

Prof. Egleston's standard of nutrition is high. He places the daily allowance of nutritive material at 700 grammes, divided as follows:—Carbohydrates, 400 grms:—Fat, 150 grms:—Proteid, 150 grms: yielding in all, 3,650 calories.

Percentage Composition of Edible Portions of Garrison Ration.

(Captain C. E. Woodruff, M. D. Asst. Surgeon, U. S. A.)

	Water.	Protein.	Fats.	Carbohy- drates.	Salts,	Energy Calories per lb.
Bacon, fat	20.0	8.00	69.5	2.5	3,080
Beans	12.6	23.10	2.0	59.2	3.1	1,615
Pork, salt and fat	12.1	0.90	82.8	4.2	3,510
Sugar, ground	2.0	97.8	0.2	1,820
Sugar, brown issue	3.0	96.5	0.5	1,795
Flour	12.5	11.00	1.0	74.9	0.5	1,644
Beef	55.0	17.10	27.0	0.9	1,460
Potatoes	78.9	2.10	0.1	17.9	1.0	375
Onions	87.9	1.4	0.3	10.1	0.6	225
Oatmeal	7.6	15.10	7.1	68.2	2.0	1,850
Cornmeal	15.0	9.20	3.8	70.6	1.4	1,645
Canned apples	83.2	0.20	0.4	13.9	0.3	315
Dried apples	25.0	0.90	1.8	71.5	1.4	1,418
Tapioca or cornstarch	2.0	97.8	0.2	1,820
Butter	10.5	1.00	85.0	0.5	3.0	3,615
Syrup	43.7	55.0	2.3	1,023
Lard	12.0	0.60	83.4	4.0	3,570
Rice	12.4	7.4	0.4	79.4	0.4	1,630
Canned corn	81.3	2.80	1.1	13.2	0.6	345
Canned tomatoes	96.0	0.80	0.4	2.5	0.3	80
Macaroni and vermicelli	13.1	9.00	0.3	76.8	0.8	1,406
Milk, fresh	14.1	0.843	0.802	1.069	0.164	418
Milk, condensed	25.0	17.00	11.0	44.00	3.0	1,595
Peas	12.3	26.70	1.7	56.40	2.9	1,565
Raisins	40.0	0.40	24.00	0.6	440
Cheese	35.0	33.00	22.0	5.00	5.0	1,600
Prunes	30.0	2.50	12.0	0.6	140
Cabbage	92.0	2.10	0.6	5.5	1.1	155
Ham	41.5	16.7	39.1	2.7	1,960
Apricots, canned	50.0	2.00	30.0	0.6	460
Barley	13.00	2.7	76.0	3.0	1,800
Chocolate	12.0	20.00	50.0	10.0	4.0	2,650
Sausage	41.2	13.80	42.8	2.2	2,065
Oysters	87.1	6.00	1.2	3.7	2.0	230
Salmon, canned	63.6	21.60	13.4	1.4	965
Crabs	15.0	1.0	526
Crackers	10.3	9.4	70.5	1,900

Church furnishes the following table showing the number of tons which it is calculated could be raised through the height of one foot by the complete combustion of a single pound of each kind of food. In the body only about a fifth of this energy would develop work, the rest going into heat production:

1 pound beef fat	raises 5,649 tons 1 foot high.
“ oatmeal	“ 2,439 “ “ “
“ gelatin	“ 2,270 “ “ “
“ lean beef	“ 885 “ “ “
“ potatoes	“ 618 “ “ “
“ milk	“ 390 “ “ “
“ ground rice	“ 2,330 “ “ “

tannic acid and alcohol, but erythro dextrin and achroodextrin are precipitated by alcohol and ether, not by tannic acid. These two dextrines do not reduce Fehlings solution and do not ferment with yeast.

3. MALTOSE:— Soluble in alcohol, insoluble in ether, reduces Fehling's solution, but not Barfoeds reagent [a weak 4 percent solution of cupric acetate to which 1 percent acetic acid is added.] does not ferment with yeast.

4. DEXTROSE:—Insoluble in alcohol and ether, reduces Fehling's as well as Barfoed's solution; ferments readily with yeast.

It is important to familiarize oneself with these reactions as it often becomes necessary to determine in cases of hyperacidity or supersecretion the degree of starch conversion.

It was formerly thought that the starch was first converted to dextrin, and this in turn was converted to sugar. According to Prof. W. H. Howell, it is believed that the starch molecule, which is quite complex, consisting of some multiple of $C_6 H_{10} O_5$ — possibly $C_6 H_{10} O_{5.20}$ — first takes up water, thereby becomes soluble [soluble starch, amylopectin], and then splits, with the formation of dextrin and maltose, and that the dextrin again undergoes the same hydrolytic process may continue under favorable conditions until only maltose is present. The difficulty at present is in isolating the different forms of dextrin that are produced. It is usually said that at least two forms occur, one of which gives a red color with iodine, and is known as erythropectin, while the other gives no color reaction with iodine, and is termed achropectin. It is pretty certain, however, that there are several forms of achropectin, and according to some observers, erythropectin also is really a mixture of dextrans with maltose in varying proportions. In accordance with the general outline of the process given above, Neumeister proposes the following schema which is useful because it gives a clear representation of one theory, but which must not be considered as satisfactorily demonstrated. [see also the section on Chemistry of the Body.]

Starch-soluble starch { Maltose.
(amylopectin.)

Achropectin. a { Maltose.

Achropectin. b { Maltose.

Erythropectin. { Maltose.

Achropectin c { Maltose.
(maltodextrin.) { Maltose.

Von Mering and Ewald have shown that in the transformation of starch into sugar by ptyalin, the greater portion is converted into maltose, only a small portion into dextrose. But the maltose formed in the stomach, is changed to dextrose in the intestine. If the amylaceous transformation proceeds normally in the mouth and stomach, after a time, within an hour at least, so much starch has been changed into achroodextrin, maltose and dextrose, that the addition of small quantities of Lugol's solution to the filtered stomach contents no longer produces any changes in color. The occurrence of a purple [erythrodextrin] or a blue color [starch] shows that the sugar transformation has been incomplete. Now this may be due either to a deficiency of ptyalin or to a rapidly increasing acidity or hyperacidity of the stomach.

Ewald says that although he tested a large number of patients for the fermentative power of saliva, he never found a saliva that could not convert starch into sugar. This too, when he tested the salivary secretion of patients with dental caries, angina, diphtheria and carcinoma of the tongue.

From the above it is evident that there must be two stages of gastric digestion, [1] an amylolytic and [2] a proteolytic. Having satisfied ourselves as regards the fate of the carbohydrates or starches, let us proceed to study proteolytic digestion or conversion of proteids, albumens, gelatins, fibrins, elastin, meat, etc. etc.

The secretion of the stomach is a complex fluid, clear, colorless, and of acid reaction, it has only $\frac{1}{2}$ per ct. solid ingredients: the amount secreted in 24 hours is about 1600 grms. Its chief constituent is hydrochloric acid which it contains in the amount of 0.1 to 0.22 per ct. (One to two per mille.) This degree of acidity is not reached at once, but gradually; at the beginning and end of stomach digestion, the percentage of HCL is considerably less. Besides the HCL gastric juice contains two unorganized ferments, Pepsin and Rennet ferment.

Hydrochloric acid acts in six different ways, all of which are of great significance for the normal progress of digestion.

1. HCL acts as an antizymotic or antiseptic, destroying pathogenic organisms and preventing abnormal fermentations. This antibacterial effect extends even into the duodenum.

2. HCL has the power to convert the proenzymes of the gas-

tric glands, (Pepsinogen and Rennet zymogen) into active ferments in a very short time, according to Langley in one minute.

3. This gastric acid possesses a certain regulatory influence on the progress of peristalsis.

4. HCL forms with the aid of pepsin—albuminous bodies into peptones, gelatin into gelatin peptone, elastine into elastin peptone. But in reality the pepsin is the main or chief agent in these transformations as the HCL can be effectively substituted by HNO_3 —phosphoric, oxalic, sulphuric, lactic and butyric acid.

5. By HCL cane sugar is changed to invert sugar. (Dextrose and Laevulose.) This property is also ascribed to a number of bacteria, that can invert cane sugar although after a longer time.

6. HCL finally is instrumental in bringing the soluble calcium and magnesium salts, introduced in the food, into the solution.

Concerning the origin and derivation of the hydrochloric acid we unfortunately have nothing but speculation. No free acid occurring in the blood or lymph, it is rational to conclude that it is produced in the secreting [oxyntic] cells of the gland ducts. It seems probable that the acid is derived from the neutral chlorides of the blood, which are in some way decomposed, the chlorine uniting with hydrogen to form HCL. The acid is secreted at the gastric mucosa whilst the base remains behind and probably passes back into the blood. This in a way explains the increased alkalinity of the blood, and the decrease of acidity of the urine after meals — the return of basic substances into the circulation naturally having such an effect. According to Heidenhain a free organic acid is secreted by the cells [oxyntic] which then decomposes the chlorides. According to Maly the HCL is the result of a reaction between the phosphates and chlorides of the blood as expressed in the following two equations.

$\text{Na H}_2\text{PO}_4$ plus Na Cl equals Na_2HPO_4 plus HCL or

3Ca Cl_2 plus $2\text{Na}_2\text{HPO}_4$ equals $\text{Ca}_3[\text{PO}_4]_2$ plus 4Na Cl plus 2HCl .

What is known thus far of the specific action of living cells, enforces the impression here as in other chemical process not yet understood and that is that vital phenomena are difficult to express in chemical formulæ.

LECTURE V.

PEPSINOGEN AND PEPSIN — RENNET AND RENNET ZymoGen — INTESTINAL DIGESTION — DUODENAL INTUBATION.

IT SHOULD not be understood that all combinations of the gastric juice with albumens are at once peptones, like the starches these proteids reach their end stage of gastric digestion by four distinct intermediate stages. These are (1) Globulin, (2) Acid albumin or Syntonin, (3) Propepton or hemialbumose, (4) Pepton. Besides forming peptones out of albumins, pepsin deprives gelatin of its property to coagulate or rather to gelatinize, and forms gelatin peptones out of it. Peptones are derived from egg, serum and plant albumens, gelatin, meat, fibrin, casein, etc.

No other mineral acids gives as good results with pepsin as HCL which can form pepsin from pepsinogen in the quickest time. It is useful to be able to test for propeptone formation. In normal digestion one hour after the test breakfast, propeptone is present only in traces, or usually is not to be detected at all, but in abnormally slow digestion it is still abundant at that period.

The best method up to present date is by means of the Biuret reaction. In this reaction a dilute solution of cupric sulphate is added to stomach contents in the cold, and a few drops of potassium hydroxide added sufficient to make the solution alkaline, an intense red color results. Cupric sulphate and KOH added to ordinary albumen and syntonin, without warming produces a bluish violet, which must be distinguished from the purple red of biuret.

The more marked the propeptone reactions are, the less the

peptone which has been formed and eventually removed from the stomach. We can approximately estimate the amount of peptone by the intensity of the biuret reaction, if we always use the same quantities of stomach contents, caustic potash and cupric sulphate and compare it with the reaction given with a peptone solution of known strength. Peptone gives the same pink, purple red-color with the biuret reaction as propeptone. If we desire to estimate the rate of proteolysis in the stomach, the biuret reaction will not permit us to distinguish between these two bodies, the only differentiation possible is by precipitation of the propeptone, in the following manner: The stomach filtrate is carefully neutralized an equal quantity of common salt solution is added and then a few drops of concentrated acetic acid. A precipitate will be propeptone which can be filtered off and weighed; any red biuret reaction after this separation must be due to peptone.

In order to determine in a given specimen of stomach contents whether the pepsin or HCL is present in too great or too small a quantity, one proceeds in the following manner:—

Pour 10 c.c. filtered stomach contents into four test tubes and number them Nos. 1.—2.—3.—4. To No. 1. nothing else is added: to No. 2. enough HCL to make a solution of 3 to 5 per mille. (This can be accomplished by adding one to two drops of official HCL U. S. Pharm. to 10 c.c. filtrate.) To No. 3, 0.2 to 0.5 grammes (gr. iii to gr. vii) of pure pepsin is added, and to No. 4. both HCL and pepsin are supplied.

A small disk of egg albumen, (which is prepared by cutting boiled egg albumen into lamellae of uniform thickness with a microtome and punching out equal circles by a corkborer) is added to each test tube and they are then put in the thermostat incubator at 100°. The rate at which the albumen is dissolved will tell us whether the filtrate was perfect in the requisite amount of pepsin and HCL, whether pepsin alone or HCL only or finally whether both were necessary. In this way we can discover which factor is at fault. In the human stomach the formation of peptone remains at a certain percentage by the removal or absorption of peptones over that amount, and also it would seem by an inhibiting influence which a certain percentage of peptone has over the proteolytic process in retarding or suspending it. As this cannot be imitated in

a test tube, *i. e.* the absorption of ready formed peptones, a seemingly delayed digestive process of egg albumen disks in the test tubes may in reality be due to a very active stomach filtrate. (Ewald.)

Rennet is the second gastric ferment which produces a light, not very cohesive coagulation of milk. This coagulation is a characteristic cake of casein floating in clear serum, more dense, not lumpy, more cohesive coagulation than produced by acids. This ferment is a constant constituent of the stomach contents just as pepsin and pepsinogen. With a complete absence of the rennet, one can with certainty conclude that the case is one of atrophy of the gastric mucosa.

The demonstration of rennet ferment is carried out in the following manner:— 10 c.c. of raw, unboiled milk are placed in the incubator with 2-5 drops of stomach filtrate. If rennet is present the characteristic milk coagulation will occur in a variable time. (1 minute to several hours, according to quantity of ferment.)

Occasionally rennet, the perfect ferment is not contained in the stomach contents whilst at the same time, rennet zymogen is present. This is demonstrated according to Hammersten by adding to the mixture just described 2 c. cm. of a concentrated solution of calcium chloride CA CL₂.

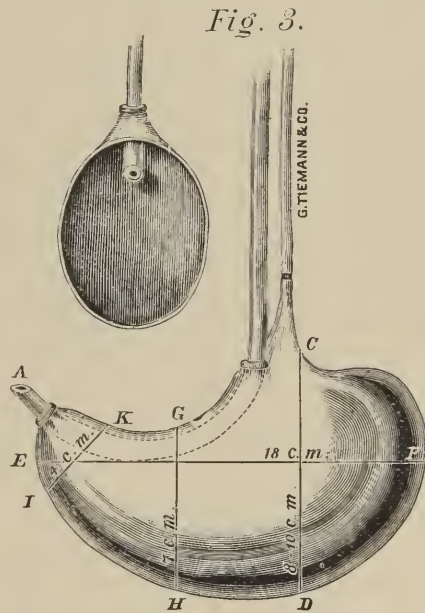
If a rennet coagulum occurs it follows that rennet zymogen is present but not the perfect ferment. For these tests, raw milk only can be used, as it coagulates 10 times as rapidly as boiled milk. Jaworski has pointed out that in cases where tests for rennet and rennet zymogen are both negative, it is advisable to try pouring a 0.3 to 0.6 hydrochloric acid solution into the stomach to see whether this HCL may not be able to awaken a secretion of rennet; this should be done especially before making the diagnosis of complete atrophy of the mucosa.

THE PHYSIOLOGY OF INTESTINAL DIGESTION.

Our knowledge of the digestive processes in the intestine, is from a physiological as well as from a pathological point of view defective, at times contradictory. Concerning gastric digestion, we are much better instructed because here the processes are simpler and material for investigation can be more easily obtained. The stomach tube supplies us without difficulty with gastric contents,

but hitherto all intestinal contents of human beings have been obtained from rare cases of intestinal fistulae, for the faeces give no information of the digestive actions in the smaller intestine, that are constant and reliable.

The earliest investigations of intestinal contents were made in 1662 by Regnier de Graaf, who made experimental fistulae into the intestinal canal of animals. It is a curious historical fact that this intestinal experiment antedated the first investigations of stomach contents which were carried on in 1752 by Reaumur. So up to the



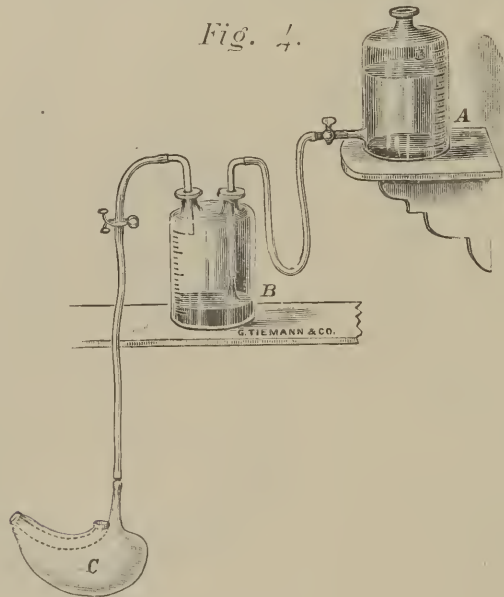
present time there was no prospect of getting a better insight into the physiology of intestinal digestion; until a method for intubating the duodenum in the living human subject was devised by myself.

This method which is described in the Johns Hopkins Hospital Medical Bulletin for April 1895, and also Boas' Archiv for Digestive Diseases, Vol. II, page 85, consists of the introduction of a thin elastic rubber bag into the stomach. This bag when folded over a tube which runs through it does not occupy as much space as an ordinary stomach tube, and has the exact shape of the human stomach

when it is distended by blowing it up within that organ, whereby it fits itself exactly to, and is closely applied to the gastric walls.

The intra-gastric bag is distended by the pressure apparatus shown in figure 4. The graduated bottle (A) is full of water and elevated above the bottle (B) which is empty and also graduated.

The stomach shaped bag, (C) when it reaches the stomach is connected with the lower empty bottle. (B) Then the stop-cock permitting the water to run from A to B is opened, and the water runs from A into B displacing the air in B which distends the bag C



within the stomach, filling it entirely. As you can observe on this bag, a guide is contained in it, running along the dotted line parallel to the lesser curvature. In this guide the duodenal tube is inserted, lubricated with oil before the bag is pushed into the stomach. This tube is provided with very thick walls, by virtue of which it is not easily kinked or bent upon itself.

The relation of the thickness of the walls to the diameter of the lumen is shown in the cross-section of figure 2. When the intra-gastric bag is blown up, it fills the stomach entirely. The duodenal tube

lies in its sheath or guide, and, on pushing onward from the mouth, it is not possible for it to go anywhere else except through the pylorus into the duodenum. In the figures it can be seen that the bag is not distended by the duodenal tube, but a separate, very small tube runs down the oesophagus, ending in the bag, serving the purpose of its distension. Both tubes together do not occupy as much space as an ordinary stomach tube.

A description of this method seemed essential because, it seems to be destined to bring our knowledge of the physiology and pathology of the intestines upon a basis of ascertained facts. Because we can at any time, thereby obtain the contents of the intestine, the gut may in any of its parts be reached with safety.

After known test meals it is possible, after they have passed from the stomach into the duodenum, to draw out samples from this part and subject them to analysis. By alternately distending any part with air or water, we will be enabled to locate the part by the percussion sound on the outside of the abdomen, and the distance it is located from the mouth can be seen from the length of tube introduced.

Small electric lamps may be introduced into the duodenum as they are into the stomach, and, the location and condition recognized by electro diaphany.

In all experiments on this subject hitherto, it has been impossible to obtain either the pancreatic or biliary secretion in a pure condition; this is due to the fact that both the pancreatic and the common gall duct empty into the descending portion of the duodenum very near each other.

Just at present I have under observation a female patient who has suffered repeated biliary colic. At times she passes small stones without giving her much pain, at least they are found in the stools without having given her any colic. She is willing to undergo an operation to be relieved. Through the comparatively thin abdominal walls I am able to feel numerous stones in the gall bladder. She consented to an attempt at intubation of the duodenum to determine whether there was any bile secreted. The duodenum was entered without difficulty and cleansed by running in and aspirating out distilled warm water. Twentyfour hours afterward the duodenum was again intubated according to my method, and washed

with 100 c.c. of warm distilled water.

On being aspirated this water was still clear but viscid and sticky, similar to a solution of egg albumen. It contained no bile pigments nor cholestrin, and was free from taurocholates and gly-cocolates. It was colorless and odorless and seemed very rich in some form of albumen. That it was pure pancreatic juice was proven by its digesting fibrin and serum albumen.

The juice obtained in this manner will digest from 85 to 95 per cent of Merck's dried serum albumen in the digestorium at 100° F. in 2 hours. The Amylolytic and fat decomposing property of the juice was determined in a similar manner. One is therefore justified in concluding that in this case the pancreatic juice was obtained almost pure as there were no bile elements contained in it; the bile being prevented from entering the duodenum by a calculus or catarrhal occlusion. As there are also pancreatic calculi, or occlusions of the duct by neoplasm or catarrhal swelling, it is conceivable that we may yet be able to obtain the bile in a pure condition, and free from pancreatic juice in the human subject.

The secretion of Brunner's and Lieberkühns glands will however always constitute an admixture of these juices.

THE PANCREAS; ITS SECRETION AND PANCREATIC DIGESTION.

In 1846 Claude Bernard made the first scientific and fundamental investigation concerning the pancreatic secretion. Later on Kühne, Bidder and Schmidt, Corvisart, Haidenhain and others enlarged these results.

Its secretion as Bernard first observed is dependant upon digestion and is a clear colorless and odorless fluid, very alkaline and so rich in albumen that it solidifies on boiling. Zawadowsky had opportunity of analyzing the normal human pancreatic secretion in a case of pancreatic fistula, which remained behind after removal of a tumor. According to his analysis it contained 86.4 percent water, 13.25 organic substances; among the latter are 9.2 proteid bodies and 0.83 extractive substances soluble in alcohol, lastly 0.34 per ct. salts.

The chyme which passes into the duodenum from the stomach comes under the influence of formed or organized and unformed

or unorganized ferments. The formed and organized ferments are represented by bacteria which bring about carbohydrate fermentation mostly in upper, bowel and proteid putrefaction, mostly in lower bowel.

The unorganized ferments are contained in the pancreatic secretion, the bile and in the succus entericus. The most important constituents of the pancreas are three ferments or enzymes. [1] An Amyolytic, [2] a Proteolytic, [3] a fat splitting ferment.

According to W. G. Halliburton and T. G. Burton, (Journal of Physiology, Vol. XX, page 106.) pancreatic juice possesses a milk precipitating substance, causing at 35°–45°c a granular precipitate in milk, but there is no solidification until the milk cools, when it sets to a coherent curd. On warming, the curd is broken up, the milk resumes its granular fluidity. The granular precipitate produced by pancreatic juice, seems according to these observers to be intermediate between casein and caseinogen.

• The amyolytic or pancreas diastase is very similar to ptyalin in its action, and changes boiled starch into maltose exceedingly rapidly at body temperature. In addition, small quantities of dextrin and grape sugar are formed; one gram of pancreatic juice from a dog will invert 3.6 grams starch into sugar. Cane sugar and inulin are not affected by it. According to Zweifel, this ferment is absent in the pancreas of new born children.

The fat splitting ferment of the pancreas, (also called steapsin) which thus far has not been obtained in a pure state, breaks up neutral fats into fatty acids and glycerine.. This process occurs very slowly however;—Berthelot found that 15 grams of pancreas secretion of the dog required at least 24 hours to break up 2 decigrams of monobutyrim completely into butyric acid and glycerin. The fatty acids formed during this transformation, combine with alkalis in the intestine to form soaps, which by emulsifying other fats assist greatly in their absorption. In the laboratory it always requires powerful mechanical action to effect an emulsion of fats, not so in the intestine, where it is evidently accomplished with great facility. That this must greatly assist in fat resorption is evident from the frequent observation that after the disease of the pancreas the feces becomes very rich in fat, which may be present in such a large amount as to congeal on the surface of the stool.

The proteolytic ferment of the pancreas has been called trypsin by Kühne. Using pancreatic juice and boiled blood fibrin, he found that it did not swell up, but that it became very fragile, and finally liquefied. As we take in all of our albumen in a boiled or roasted state, which becomes peptones in the stomach and not soluble albumen, the question has arisen—where do we derive our soluble native albumen? This is obtained from pancreatic trypsin digestion of boiled albuminous bodies, which changes them to albumen soluble in water or at least in weak saline solution from which they can be precipitated by heat. The proteolytic action of trypsin, takes place in an alkaline or neutral medium only.

Among the bodies formed from albumens and proteids under the influence of trypsin are a glolulin that is insoluble in water, hemi and anti-pepton, leucin, tyrosin and asparaginic acid. Indol, which is found in the jejunum is a product of bacterial action on albumens. A chromogenic body has been described by Tiedeman and Gmelin which has received the name tryptophan, it is a result of extensive albumen decomposition. Trypsin, then to sum up, changes proteids to peptones and soluble albumens, casein to casein-peptones, gelatine to gelatoses and gelatin-pepton, and, elastin to elastoses and elastin-peptones.

In animals that have been deprived of their pancreas by operation only 44% of proteid, 57% to 70% of carbohydrates and no fats at all were absorbed, although four-fifths of the fats were split up into fatty acid and glycerin.

LECTURE VI.

THE BILE — THE SUCCUS ENTERICUS — INTESTINAL FERMENTATION — PUTREFACTION — FORMED OR ORGANIZED FERMENTS.

IT IS KNOWN at present that the bile exerts no chemical effects upon the food materials, nevertheless its presence in the duodenal chyme is significant on account of its alkaline reaction and its effect on the mucous membrane. The most important function of the bile is the excretion of metabolic products that cannot be utilized.

The contents of the gall-bladder represents a concentrated secretion, therefore our knowledge of the physiological action of the bile depends upon the discharge of biliary fistula. The bile is a golden yellow, at times olive brown secretion; it is never of a green color, but generally very mucoid and stringy. Its alkaline reaction is due mainly to carbonates and phosphates. The quantity poured into the intestine is largest in the first hour after food is taken.

Albumens increase, fats diminish this quantity, whilst sugar and carbohydrates appear to exert no influence. (Voit.) The quantity secreted in 24 hours averages 5–600 c. cm. (Ranke, Wittich,

Hammarsten.) The quantitative analyses of Hammarsten have given the following results:

Solid materials	1.62	—	3.52
Water	96.47	—	98.37
Mucin and coloring matter	0.27	—	0.91
Compounds of bile acids & alkalies	0.26	—	1.82
Taurocholate	0.052	—	0.203
Glycocholate	0.204	—	1.61
Fatty acids	0.024	—	0.136
Cholesterin	0.048	—	0.160
Lecithin	0.048	—	0.065
Fat	0.061	—	0.095
Soluble salts	0.676	—	0.887
Insoluble salts	0.020	—	0.049

At times a diastatic ferment is present in the bile, but it is not a specific constituent (Neumeister) but appears in the bile like the diastatic ferment appears in the urine; it seems to be identical with the ptyalin zymogen of the urine.

When the bile is prevented from entering the intestine at all, albumens, gelatines and carbohydrates are absorbed in a normal manner. (Voit and J. Munk.) But the digestion of fats is very seriously interfered with; a normal animal resorbes 99% of fats if not more than 150 to 200 gr. are given *i. e.* only 1% appears in the feces, but on producing an experimental fistula conducting the bile outward, 60% of the fats are not utilized. (Voit.) The subjoining is a synopsis of the uses and functions of the bile:

1. Fats are brought into a fine, permanent emulsion by bile just as by pancreatic juice.
2. Bile assists the fat splitting effect of pancreatic juice. (Nencki.) Without bile only 61% of tribenzoicin were decomposed by pancreatic juice, with bile the total amount.
3. By its alkalinity it necessitates the formation of soaps.
4. Bile dissolves fats in minute quantities.
5. Bile dissolves the saponified alkaline bases which are insoluble in the juices of the intestines.
6. Animal membranes moistened with bile are more permeable to emulsified fats than membranes moistened with water. (v. Wisting Heidenhain.

7. Bile is a stimulant to the intestinal epithelial cells, incites their proper functioning and maintains it. (Röhmnn.)

8. It is claimed that albuminous bodies and pepsin dissolved in the chyme are precipitated as a resinous sticky deposit which adheres better to the duodenal wall and effects a better utilization of the albuminates thereby.

9. An inhibitory influence over putrefaction is ascribed to bile (Maly and Emmerich.)

10. An influence favoring an increase of the peristalsis of the intestine. (Röhmnn.)

THE SUCCUS ENTERICUS.

The succus entericus is a secretion of the crypts of Lieberkühn and was first studied in man by Demant after a herniotomy. This secretion has the color of light rhinewhine and is very strongly alkaline owing to a percentage of 1.5% carbonate of sodium. The principal constituents are albumens and mucin. It contains also ptyalin and an inverting ferment, and has no effect on albumens and fats—its purpose seems to be probably that of a neutralizer of the acids originating from fermentation of carbohydrates. For the onward movement of the bowel contents, its excess of mucin may be instrumental.

THE FORMED OR ORGANIZED FERMENTS. — (BACTERIA.)

Proteids, carbohydrates and fats are subject to decomposition in the intestines by bacteria. Fats are not decomposed to any considerable extent in the lower intestinal sections (Nencki and Blank) but a small fraction is split up into glycerin and fatty acids.

A greater interest attaches itself to the fermentation of carbohydrates, occurring in the upper small intestine principally and leading to the formation of acetic, lactic, butyric acid and alcohols, carbonic acid and hydrogen. It is not known how much of the carbohydrates is decomposed in this manner.

The putrefaction of proteids, caused by certain bacteria of the lower bowel only occurs in an alkaline medium. The first products of this putrefaction are the identical bodies which are formed during pancreatic digestion—viz: albumoses, peptones, amido acids and ammonia, but then the putrefaction goes still further, tyrosin is

formed, and from this, through a series of complex, oxyacids—the product phenol (carbolic acid) is reached, which may yield phenyl propionic and phenylacetic acids. A second variety of aromatic substances, not derived from tyrosin, is represented by indol, skatol and skatol carbonic acid, finally leucin and ammonia salts of capronic, valerianic and butyric acids. The gases formed are carbonic acid gas, hydrogen, hydrogen sulphide and methylmercaptan.

We cannot measure the intensity of carbohydrate fermentation, but the aromatic end-products of proteid putrefaction can be quantitatively estimated by the combined and ethereal sulphates occurring in the urine.

The number of bacteria increases from the duodenum on downward until they become enormously prolific in the colon. They differ qualitatively also; in the small intestine, Gessner found a prevalence of the bacterium lactis aerogenes and streptococcus pyogenes, the colon bacillus was present but insignificant in numbers. In the colon however, the reverse was the case. — It was formerly an accepted view principally defended by Pasteur, that the intestinal bacteria were absolutely indispensable for digestion and therefore for the nutrition of the individual. From this view we have returned to what seems a more logical belief based on observations of Escherich who held that bacteria contribute very little to the nutrition of the infant, as they do not effect casein and fats, but only sugar of milk. [Turning it into lactic and carbonic acid and hydrogen.] The work of Nuttal and Thierfelder shows that guinea-pigs can live on absolutely sterile food.

Macfadyen, Nencki and Sieber arrived at a similar conclusion in their now classical observations on adults. [Archiv f. exp. Pathology, Band xxviii, 1891. One of their objects of study was a female with a fistula that opened the small intestine on the external abdominal wall, just at the end of the ileum. The entire colon was therefore excluded from the digestive act, as all proteid putrefaction occurs in the colon, this case presented a chance to study the absence of products of albuminous putrefaction and its effects.

They concluded that bacteria are not at all essential to digestion, as their patient was very healthy without proteid putrefaction. They declare that the bacterial fermentation of carbohydrates in the small bowel as detrimental rather than advantageous inasmuch as the

bacteria live at the expense of the ingested carbohydrates, therefore a corresponding amount of food is lost to the organism.

Our knowledge of the bacterial activity in the intestines, though much enriched by valuable researches in the last decade is according to my opinion in its infancy. So also our knowledge of the pathogenic significance of intestinal bacteria. There is undoubtedly a kind of interaction and correlation between digestive ferments and juices on the one hand and bacteria on the other, or even between bacteria and bacteria, or between the products of bacterial metabolism. For instance, Metschnikoff has demonstrated that the multiplication of the cholera vibrio is much advanced by the presence of torulæ and sarcinæ in the intestines.

It is conceivable that bacteria wage war upon each other as well as upon our cells, and, that we are benefited by this mutual self-destruction of our parasitic inhabitants. It is conceivable also that that they fall preys to the poisonous metabolic products of their own or other species of bacteria. Certain very decomposable food as cheese, that was rich in germs, has been found by competent observers to reduce the amount of indican and the ethereal sulphates in the urine.

The human stomach must not be regarded as an organ that can absolutely sterilize all food. The spores being more resistant to HCl than the fully developed bacteria themselves, pass through the stomach uninjured. Miller assumes that at the height of digestion only, when the amount of HCL is greatest, the less resistant bacteria are killed. Bunge some years ago announced that the sole object of the Hcl was one of sterilization. It is undeniable from recent investigations, that the human stomach is at no time free from germs. Captain and Morau found them at the height of digestion. Abelous found them in his own stomach when it was perfectly empty. Miller demonstrated that the mouth contains large numbers of microbes, in one unclean individual he estimated the numbers of mouth bacteria at 1 140 000 000. Of 25 different varieties occurring in the mouth this observer was able to demonstrate 12 of the same in the feces. Nevertheless the mouth bacteria according to Lucksdorff constitute only 3% whilst those entering through the food constitute 97% of the bacteria of the intestine. There is no autochthonous vegetation of bacteria in the intestine, they are all introduced from the mouth

or in the food, or reach there by way of the circulation. (Autochthonous bacteria means—such as are formed where they are found.)

From observations made up to the present time it seems probable, that catarrhal and other inflammatory diseases of the intestinal mucosa are not produced by specific, constantly recurring microbes: but that a large variety and many kinds of bacteria are capable of producing these diseases under conditions which are thus far not perfectly understood.

It appears furthermore that the same bacterium may at one time be perfectly harmless, or it may cause a light, trivial affection or thirdly a very serious disturbance. This is the case with the bacterium coli communis, which is borne without detriment by the majority of mankind, but occasionally it is demonstrated as the producer of colitis, Dysentery and Cholera nostras.

The manifold forms of the catarrhal inflammations are explicable by the fact that the intestinal flora is very manifold also. These same bacteria are factors in the etiology of diseases of the peritoneum and of all organs that are in connection with the intestines. Even remote organs, not in anatomical connection with the bowel are not safe from their invasion.

They are known to gain entrance into the blood and lymphatic channels through losses of substance in the intestinal mucosa. The experiments of Posner and Lewin have taught us that even without such portals of entry they seem to be able to pass through the bowel wall in masses and threaten the organism. Great harm can be done to the general organism, and to special organs in particular not from this invasion only, but also from absorption of the soluble products of bacterial metabolism and of food decomposition.

This condition of self-poisoning from toxic substances in the individuals own intestinal canal is spoken of as intestinal autointoxication. Not all autointoxications are of intestinal origin — diabetes mellitus for instance is an autointoxication by grape sugar which is in this case a product of disturbed metabolism and does not originate from the digestive canal.

The dangers which threaten the general organism from the intestinal bacteria have given rise to many efforts to sterilize the digestive tract by means of so called antiseptics. Most of the agents used for this purpose — (Calomel, Salol, Naphthalin, Beta-

naphthol, Bismuth, Creosote, Bismuth, Salicylate) are themselves toxic and in doses sufficiently large to reduce the number of bacteria to any considerable extent they are harmful to the body. The putrefaction of proteids as measured by combined and ethereal sulphates in the urine can only be temporarily diminished by this method.

Intestinal disinfection is therefore an unsolved problem. Efforts in this direction should still be encouraged, because we may be able thereby to attenuate the pathogenic inhabitants of our insides and render them less virulent. The best disinfectant of the human intestine is its normal action, and the best way to control putrefaction is by selection of adapted, appropriate diet, fresh air, moderate exercise, good sleep, pure water, avoidance of overeating, overdrinking and of chemicals.

LECTURE VII.

THE EFFECTS OF CONTEMPORANEOUS ACTION OF SEVERAL DIGESTIVE SECRETIONS — QUALITATIVE AND QUANTITATIVE METHODS FOR TESTING THE MOTOR, SECRETORY AND ABSORPTIVE FUNCTIONS OF THE STOMACH.

WHEN THE GASTRIC chyme enters the duodenum, the albuminoid and proteid foods appear partly as syntonin, albumoses and peptones, and partly unchanged. The carbohydrates appear either as erythrodextrin, achroodextrin or maltoses, and partly unchanged. The fats are unchanged; rarely are they found split up—so that one can detect traces of fatty acids.

Water, according to the interesting investigations of v. Mehring, is absorbed only in very small quantities from the stomach; it appears that fully 99% of all water taken into the stomach is passed into the

duodenum;—alcohol and whatever is in solution in it, is absorbed readily. Grape, milk and cane sugar, also maltose are absorbed in moderate amounts when they are in aqueous solution; when they are in alcoholic solution, larger amounts are absorbed. Dextrin and pepton are also taken up from the stomach but in smaller quantities than sugar. The amount of the substances resorbed increases with the concentration of the solution. Simultaneous with this resorption a more or less active excretion of water occurs into the stomach. This excretion of water into the stomach increases or diminishes as the quantity of substances resorbed or taken up increases or diminishes.

Excretion of water occurs even when no HCL is demonstrable in the stomach. The chyme, as it enters the duodenum then, still contains all of its water but is minus some of the peptones, dextrins, sugars and alcohols. It is more or less acid from free HCl, when the bile acts on this acid chyme a resinous flocculent precipitate is deposited from it on the walls of the duodenum, at the same time a finely granular cloudiness occurs. The resinous deposit consists of bile acids and syntonin (Hammarsten), and the granular opacity is due also to bile acids and small amounts of peptone.

Excess of bile may redissolve these precipitates so that they can not at times be found in animals killed at the height of digestion. The digestion by pepsin is checked by the complete neutralization of HCl by pancreatic juice, bile and succus entericus. If any precipitation occurs as stated, pepsin is also thrown down and resorbed again. The bile does not disturb the proteolytic power of pancreatic juice (Claude Bernard.) Boas and myself have shown that duodenal chyme will digest 81% serum albumin in 3 hours at 40°C. its alkalinity was 0.8% Na_2CO_3 . He also showed that this duodenal chyme converted 25% starch into maltose in 3 hours and that it produced 12.1% fatty acids from neutral olive oil in 3 hours. Boas obtained his mixture of bile pancreatic juice and succus entericus, from a patient who had most probably a duodenal stenosis and vomited this chyme frequently. In my experiments, the duodenal chyme was obtained by my method of intubation of the duodenum.

It was found in my experiments that the duodenal juices, when filtered digested 85% to 95% of Merck's dried serum albumen in 3 hours at 40°C. My results with starch conversion show that the

filtered duodenal juices will digest 42% of starches or rather convert them into maltose which is considerably in excess of the figures obtained by Boas. The fat splitting effect observed by me in this juice was again more near the result of Boas, for in my experiments 15.3% of fatty acids were obtained from neutral olive oil. In a case of billiary calculi, I have been able to obtain the pancreatic juice free from bile as the bile ducts must have been stenosed; either by a small calculus or a bit of thickened bile and mucous mixed.

The fat splitting effect of pancreatic juice is improved by the presence of bile, as is also the amyolytic action of amylopsin.

(Demonstration)—You have an opportunity here of studying the action of pancreatic juice on neutral olive oil 10 c.c., and also on starch 10 c.g. in distilled water. This pancreatic juice was obtained from a dog in pure state by putting a canula in the pancreatic duct. I will arrange two test tubes, each with neutral oil, but add 10 c.c. of bile to one only; the other contains simply pancreatic juice; then 2 more test tubes are arranged, each with a known quantity of starch; say that 10 c.g. of starch are added to each, in one is contained only pure pancreatic juice, in the other pancreatic juice with bile. Now we will place these four test tubes; two with pancreatic juice only and neutral oil and starch respectively, and two with pancreatic juice plus bile and oil and starch in each respectively in the incubator at 40°C. for 3 hours, and, tomorrow you can determine the amount of oil split up in the test tubes containing it and convince yourselves that pancreatic juice plus bile will split up more fat than without it, and that it will convert more starch into maltose with bile than without it. (Martin and Williams.)

Trypsin has no effect on pepsin, but in an acid solution, pepsin is claimed to check the action of trypsin. (Kühne, Langley, Ewald and Baginsky.) The ferment action of bacteria in the small intestine is limited to the carbohydrates. Discharges of food from fistula of the small intestine show no foetid decomposition of albuminoids (Ewald and Nencki.) The absence of proteid putrefaction in the small intestine is probably due to the rapid downward movement of the food mass in this bowel portion and to the acid condition.

Carbohydrate fermentation yields mainly lactic acid, aethyl alcohol, carbon dioxide and hydrogen. Macfadyen, Nencki and Sieber found that the chyme that passed over into the large intes-

tine, the coecum, from the ileum to be 550 grs. with 4.9% solid residue in case the chyme was of a very thin consistence, and 232 grs. with 11.23% solid residue if the chyme was very condensed. Both of these figures are the amounts passing in 24 hours. the shortest time in which food passed into the coecum after it was swallowed was 2 hours, the longest period $5\frac{1}{4}$ hours.

The reaction expressed in acetic acid was equal to 1 pro mille; the acidity is considered to be due to acetic acid, as the lactic acid and the HCL is neutralized by the succus entericus. This chyme contained 1% albumen, pepton mucin, also dextrin and sugar, also lactic acid, sarcolactic acid and traces of fatty acids; it contained no leucin, tyrosin, urobilin, nor ammonia.

This chyme contained none of the characteristic products of albuminoid decomposition. Jakorowski's investigations on the contents of the large intestines that were discharged from a fistula in the ascending colon, showed that the daily fecal discharge of 150 – 200 gr. was decidedly alkaline and contained the products of proteid and albuminoid decomposition;—viz urobilin, skatol, phenol, oxyacids, ammonia, leucin, cadaverin, aethyl and butyl alcohol sulphuretted hydrogen and methyl mercaptan.

In view of the fact that the putrefaction of albuminoids and proteids occurs mainly in the colon, it is of interest to know how much of this class of food substances is left for the colon and how much is digested in the small intestine. Nenki found that when the food contained 70.74 grs. albumen which represents 10.602 grs. nitrogen—the amount of solid material discharged from a colon fistula in 24 hours was 26.5 grs. with 1.61 grs. nitrogen which represented 10.06 grs. albumen. From this it is evident that 14.25 % or in other terms only one seventh of the total albumen is left for the digestion in the colon, and that 85.75 % are resorbed from the stomach and small intestine.

The intensity of putrefaction in the colon depends upon four factors: (1) The amount of decomposable albuminoid material ingested. (2) The duration of their retention in the colon. (3) The vigor and tonicity of the intestinal peristalsis and lastly (4) the chemical reaction for a very strongly acid reaction due to free acids inhibits putrefaction.

Bile assists in this inhibition. Hirschler has demonstrated that

carbohydrates suppress putrefaction considerably which is due to the lactic, butyric, acetic, and carbonic acid caused by their fermentation. Albumen and pepton are absent from the contents of the rectum (feces) but are present in typhoid fever (v Yaksch) pepton is found also in all diseases that may produce pus in the evacuations for instance dysentery, tuberculous intestinal ulcers, perforation, peritonitis, hepatic cirrhosis and carcinoma.

A very important inquiry is that into the ultimate fate of the digestive ferments; do they pass through the entire intestinal tract? are they absorbed or are they decomposed or do they appear in their active form in the feces. This question is a very difficult one to solve, as our only methods of detecting pepsin, trypsin, and ptyalin for instance is by their digestive activity. In all experiments of this kind the feces must be first sterilized by saturated solutions of Thymol, before using this it is well to exclude the action of peptonizing bacteria by removal of these by filtering through a Pasteur filter.

If we found in the glycerin extract of the sterilized feces a substance which would dissolve boiled egg albumen in a solution of 0.2% HCL we should be justified in concluding that it was pepsin. If it did not digest in HCL but in a .1% solution of sodium carbonate it would probably be trypsin.

For the demonstration of a diastatic ferment a dilute solution of starch is brought into the incubator with about 5 c.c. of glycerin extract of sterilized feces. After a few hours the HCL and soda solution of the boiled albumen is tested for pepton by the biuret reaction and the diastatic test tube is tested with a dilute solution iodide of potassium, if the starch is unchanged the solution will be changed to blue, if not, the color will be brown or yellow.

The digestive action of the succus entericus which according to Grützner has a weak fibrin dissolving property, does not extend to the albumens and therefore it will not confuse the result stated above as pertaining to pepsin and trypsin.

The chief digestive action of succus entericus is on the carbohydrates. If pepton occurs in the stools it is in my opinion a product either of pepsin or trypsin digestion, not of bacterial origin. Undoubtedly there are proteolytic bacteria, for instance the bacillus subtilis of Ehrenberg the proteus vulgaris of Hauser, the bacillus putrificus coli of Bienstock and the bacillus liquefaciens ilei Macfadyen

Nencki and Sieber, all of which exist ordinarily in the human intestine, and their first products of action on albumens are the same as occur in normal pancreatic digestion — viz. Albumoses peptones amido acids and ammonia; but then the action continues uninterruptedly to the production of decomposition bodies stated in previous lecture. The bacterial produce of peptone is probably of no use to the organism in which it occurs, as bacteria do not make peptone for any philanthropic reason, but because it is a first stage to proteid putrefaction and possibly because these proteolytic parasites need peptone for their own existence.

The remote possibility that bacteria could produce peptone in the colon (feces) might be excluded by the fact that after sterilization of the feces by a saturated solution of thymol: peptone will in some cases still be produced when above tests are made — more often will this be due to pepsin as it occurs only in an acid medium during the testing, but rarely it is due to trypsin which is present only in the stools where they have traversed the intestines rapidly.

Starch inverting ferments are present in the saliva, pancreatic juice and succus entericus, hence if such a ferment appears in the feces it is impossible to conclude its source.

Amylopsin and steapsin have as such not been demonstrated in normal feces. Pepsin and rennet occur in normal feces. Personally I have found a proteid dissolving ferment in the stool which acted in a 1% solution of carbonate of sodium only, and was studied in a case of complete atrophy of the gastric mucosa, with total absence of HCl, pepsin and rennet, and also the proenzymes of these ferments. In the wash water bits of mucosa were found that proved the absolute destruction of the glandular apparatus of the stomach.

It is probable that this ferment was trypsin, there was a moderate gastrectasia, but otherwise no anatomical defect observable.

The stools were not diarrhoeic. Escherich's assertion that the colon bacteria do not live upon the food introduced; as according to his opinion, there is no digestible food left there under normal conditions, but that they live upon the secretions of the walls of the colon is certainly — if this statement of his view is correct — (Mannaberg in Nothnagel's *Erkrankungen des Darms*, p. 38.) erroneous.

The conception of some writers on this subject, that food materials are completely used up in the digestive tube, is not proven

by actual fact. Even meat when eaten in a most digestible form is found undigested to a small extent in the evacuations. It is therefore more than probable that the colon bacteria live at the expense of the ingested proteid food.

Having thus far reviewed the physiology anatomy and in parts the pathology of food digestion in general, let us now return to the special pathology of the functions of the stomach as a preparation for a better comprehension of its diseased states.

QUALITATIVE AND QUANTITATIVE METHODS FOR TESTING THE MOTOR, SECRETORY AND ABSORPTIVE FUNCTIONS OF THE STOMACH.

The motor or peristaltic function is the most important one, because a man may be able to live without the secretory and resorptive function of his stomach as the intestinal digestion and secretion would suffice for the conversion of carbohydrates and proteolysis and one depends upon the small intestine for the digestion and absorption of fats altogether. So that even in the total absence of gastric resorption and the falling away of secretion of HCL, pepsin and rennet ferment; life could be maintained.

But if the motor function is interfered with the food would remain in the stomach, and accumulate. If a normal gastric juice were even possible when the peristalsis is paralyzed, the food could be only partly digested. Carbohydrates and fats would not be digested. When the limit of distention was reached the food would be ejected (Pyloric Stenosis Gastrectasia.)

In all cases of inhibition or loss of motor power the secretory power is seriously disturbed or may cease absolutely so also the resorptive power. Many cases of total absence of gastric secretions have been reported — (in fact I have shown you a colored patient in the clinic in whom this is the case and yet he is gaining weight on carefully selected diet) in patients whose body weight continued normal and their general health unimpaired. The stomach has been removed experimentally in dogs and the animals continued to thrive without it.

There have been up to very recently six different methods proposed for determining the motor functions of the human stomach.

The methods of Leube, Ewald and Sievers, Klemperer, Fleischer, Einhorn and Hemmeter.

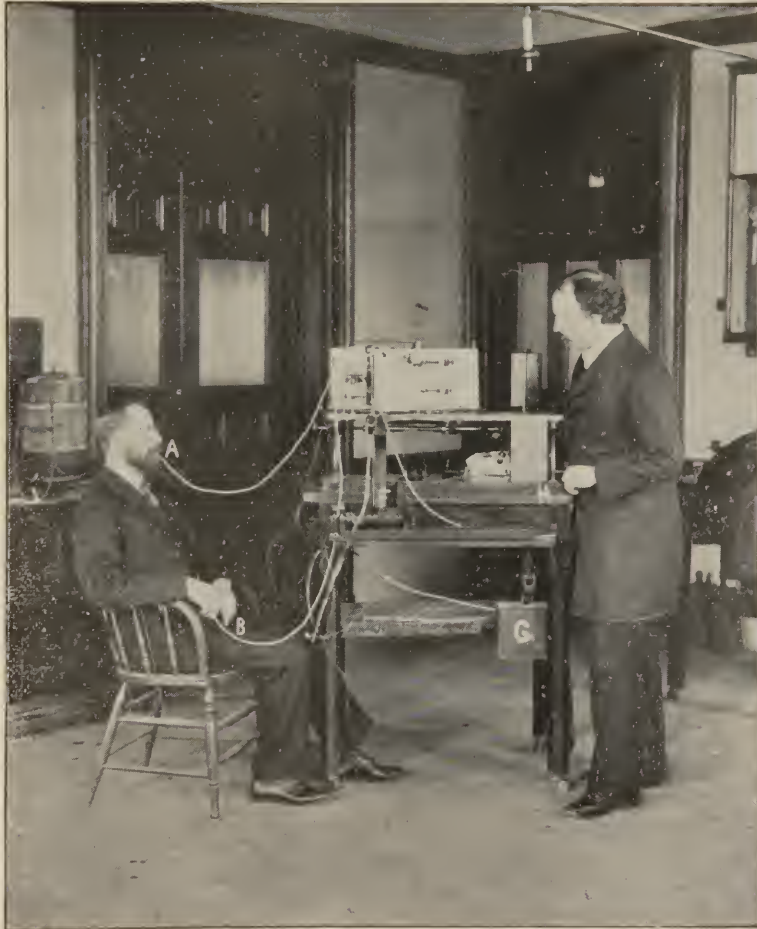
Leube's method of estimating the duration of digestion *i. e.* to determine after a definite average time of six to seven hours after a meal of 50 grs. bread – 200 grs. beefsteak and a glass of water, or two hours after an Ewald test breakfast whether solid contents were still to be found in the stomach,— will serve the practitioner with a simple and ready method which follows naturally in the line of drawing test meals from the stomach, it is however subject to too many physiological variations to permit of accurate deductions.

Ewald has proposed the use of salol which according to Nenki is not decomposed by acids (in the stomach), but is converted by the alkaline juices of the duodenum into salicylic acid and phenol. He found in connection with Sievers that the appearance of salicyluric acid the product of the decomposition of the salol in the urine would indicate that the salol had actually passed out of the stomach.

Normally, salicyluric acid will appear in the urine in from 40 to 75 minutes after taking one gramme of salol. Delay in its appearance will indicate a retardation in the passage of food into the intestines.

Salicyluric acid is recognized in the urine by the violet color produced on the addition of neutral ferric chloride solution. You will detect that this method necessitates frequent urination of the patient, every five minutes at least, otherwise the result will not be accurate.

Brunner, Riegel and Eichhorst found that the time in which the reaction occurred in the healthy individual varied from 40 minutes to two hours. This was to be anticipated as the period that a test meal may remain in the stomach may vary normally between 2 and 3 hours.



Subject and experimenter working at kymograph.

LECTURE VIII.

METHODS FOR TESTING THE MOTOR FUNCTIONS OF THE STOMACH. (Continued.)

AS EWALD'S SALOL test is not applicable in private practice because of the frequent micturition that is necessary, it is impossible to examine females, and, also because the excretion of salicyluric acid depends upon the changing energy of the heart's action, intra-arterial pressure, the amount of water in the blood and the changeable function of the kidneys themselves.

Huber improved this method somewhat by ascertaining that salicyluric acid disappears from the urine after administration of salol to healthy persons, in 24 hours, but in patients with impaired gastric peristalsis, the reaction continues to be distinct much longer sometimes for 48 hours. According to Fleischer and Hecker the duration of excretion of Potassium iodide in the urine of healthy individuals varies from 29 to 55 hours, of sodium salicylate from 21 to 29 hours and in cardiac and nephritic patients this may vary from 80 to 96 hours.—It is evident that methods of such a variable character are not satisfactory for exact research; nor even on account of the great loss of time, of much value for comparative tests.

Klemperer's method consists of the introduction of 100 grs. of neutral olive oil into the perfectly clean stomach after lavage, through a stomach tube. Oil or fatty acids which are formed in traces are not absorbed from the stomach. After 2 hours, all oil yet remaining is washed out by repeated lavage, dissolved in ether and weighed after removal of the ether by distillation. In the normal subject

Klemperer could find but 20 – 30 grs. of oil, the remaining 70 – 80 grs. had passed into the intestine. If larger amounts are found, for instance 50g, 60g or more, they are according to Klemperer, an evidence of motor insufficiency. This method requires very much time and skilled chemical analysis, and, is also open to the same objection as that of Leube.

Fleischer (Spez. Path. u. Therap. d. Magen u. Darm. Krankh. page 791.) has proposed a method to determine the gastric peristalsis by giving 0.1 gr. iodoform in a gelatine capsule during meals; this compound does not decompose in acid media, but does break down in the alkaline juices of the duodenum, and one of its resultants is potassium iodide which can be tested in saliva by starch paper, which, when dipped into the saliva, colors blue on being touched with a drop of fuming nitric acid. Naturally the potassium iodide can also be detected in the urine, but the fact which gives this method the preference over Ewald's salol test is that the KI can be detected in the saliva.

It is quite a variable experiment as I have discovered; In 23 cases in which I have tried it, the reaction coloring the starch paper first occurred just 1 hour after the meal in 12 cases, in 6 cases it occurred first in 1 hour and 20 – 22 minutes, in 2 cases in 1 hour and 41 minutes, and, in 1 case in 2 hours;—in 2 cases it took 2½ hours to demonstrate KI in the mouth after giving 0.1 iodoform.

These were cases in which the gastric secretions were known to be normal. The time of the appearance of the first red and the subsequent blue coloring of the starch paper was carefully noted. Fleischer states that after a test breakfast the reaction in the saliva should occur in from 55 to 105 minutes which is still a considerable margin for variations; too great for practical purposes.

Nevertheless the method is interesting and with exactly known meals, might be available for hospital work.

In Leube's, Ewald's, Siever's, Klemperer's and Fleischer's methods it will be observed that the gastric motility was determined by something that was administered (salol, iodoform and food.) or poured into the stomach, (oil) and by the absorption of the product of breaking down in the alkaline duodenum and its subsequent appearance in the secretions and excretions, (pot. iod. in the saliva and salicyluric acid in the urine) — an expression in terms of time was



Patient with intragastric bag within stomach and pneumograph in place, both connected with kymograph.

arrived at, to denote the intensity of the gastric peristalsis.

In two methods the expression is derived from the quantity of oil or food retained in the stomach after two hours, but also here the result depended upon the passage of something into the duodenum. In all of these methods therefore the fundamental idea is, the rate of expulsion of gastric contents into the duodenum, as if that were the only object of the motor functions of the stomach.

It is probable that this, which is only a part of the purpose of the gastric peristalsis, was so much dwelt upon because it offered the most expedient means for experimenting, and, a greater possibility of solution of the problem. However, a second and most important purpose of the gastric peristalsis, and one, concerning which none of the methods referred to, thus far, can instruct us, is the moving about of the ingesta within the stomach (1) so that they may be made into a more homogeneous mass, (2) so that they may be brought into thorough contact with the gastric juice, and (3) to stimulate the secretion of this juice by the mechanical irritation of the walls of the organ.

These in addition to expelling the chyme are the purposes of the motor function. The secretion of the gastric glands is not only stimulated by the mechanical irritation of the stomach walls during peristalsis, but by the contraction of a liberal supply of muscular fibers, which arise from the muscularis mucosæ, and are spun around the fundus or bases of the gland tubules, the glands are no doubt themselves contracted, and their contents expelled.

In some of the batrachians this contraction of the gastric gland tubules by electric stimulation is visible under the microscope.

Dr Max Einhorn, the pioneer worker in the pathology and treatment of digestive diseases in this country, has described in the New York Medical Journal Sept. 15 1894 an instrument which records the gastric movements by dots on a narrow piece of paper.

This apparatus consists of a ball about $\frac{5}{8}$ of an inch (14 millimetres) in diameter, which is made up of two hollow metallic hemispheres which are screwed together. Within this is contained a second smaller ball, which is attached to the upper hemisphere by a non-conductor so that it is insulated from it.

The central smaller ball bristles with small metallic spikes radiate in all directions, from the center to the inside of the two

hemispheres, but not touching them.

A tiny platinum sphere, completes the interior of this apparatus, it lies within the larger round capsule and moves about knocking at the spikes. When it does so, it completes an electric circuit between the outer hemispheres, and the spikes of the central ball; — for two insulated wires, one connected with the hollow ball, the other with the spiked ball run up in a very fine, thin, rubber tube and are connected with the two poles of an electric battery. On connecting the ball with another part of the apparatus, the "ticker," (very much like the instrument used at the stock exchanges for reporting the variations in stock by telegraph.) — each motion of it will be recorded by lines or dots on the paper. The ball is swallowed and brought into the stomach by the aid of a few swallows of water. It must be born in mind that the paper records the motions of the ball only; this does not mean that it records every motion of the gastric peristalsis.

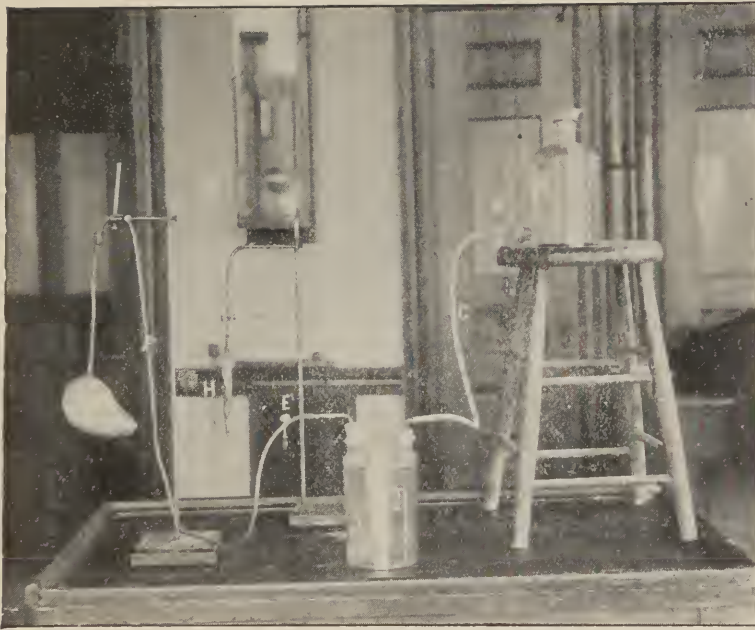
In animals upon which I experimented at the biological laboratory of the Johns Hopkins University, a rubber, stomach shaped bag was fitted exactly to the interior of the animal's stomach, and, connected with a manometer on the Ludwig kymographion. Records were taken with the animal's abdomen intact and compared with those with the abdomen opened; so that the gastric peristalsis could be viewed by the experimenter.

The physiological peristalsis is essentially the same whether the animal's stomach is normally contained with the abdomen or exposed to view.

In my experiments the animals were placed in a large metal case with a glass top, underneath the animal holder, about 2 inches of water was contained in the bottom of the case, which was kept at any desired temperature by a number of Bunsen burners beneath the case. Thermometers were suspended from different parts of the case to keep watch on the temperature, for it is most essential that after an animal's abdomen has been opened, it should be kept at a constant temperature by moist steam; this also insures the viscera against becoming too dry.

In a similar manner, Ludwig and H. Newell Martin studied the physiology of the mammalian heart;—Schatz conducted his fundamental investigations on the contractions of the uterus; — Engel-

PLATE III



The apparatus, not including kymograph. G, intragastric bag distended; F, the œsophageal tube attached to it; H, intragastric bag collapsed in the shape it is introduced, A, pressure bottle elevated and filled with water and graduated; B, stopcock; D, lower graduated bottle, empty at first. The bag is distended after it is swallowed by connecting it at E with D; the stopcock B is turned on, and the water then runs from A to D, displacing the air in D and forcing it into the bag. Both bottles being graduated, the amount of air in G is always known and can be utilized as an indication of the gastric capacity.



mann, his pioneer work on the contraction of the involuntary muscle fibers of the ureter;— Phlūger and Haidenhain have done similar accurate work on excised organs, the results have been repeatedly confirmed by other competent investigators.

These epoch making experimentations are mentioned to assure you that experiments conducted on organs isolated either entirely (Martin – Ludwig – Engelmann) or partially (Schatz – Phlūger) are capable of giving perfectly physiological contractions or peristalsis which differ in nowise from the perfectly normal ones.

It is frequently urged that these experiments on account of the operations and the anaesthesia necessary, do not present perfectly physiological conditions, and that therefore the deductions made therefrom are not logical nor represent the true state of normal functioning.

It is undeniable that we never get at the absolutely exact normal functioning of an organ, the stomach for instance during an experiment, as ether and chloroform have an inhibiting effect on the gastric peristalsis. But we are enabled to produce unconsciousness of the animal after a brief ether narcosis by brain compression, and then the gastric peristalsis continues perfectly normal.

The stomach of the rabbit will show normal peristalsis after complete excision and suspension on a hook or clamp in a warm moist chamber. What brought me to the idea of using an intra-gastric thin rubber bag to record the peristalsis after many attempts with a small spherical bag, that did not exactly fill out the entire lumen of the stomach, was the repeated observation that the small round bag, such as Prof. Moritz of Munich used, did not record every peristaltic movement that was visible to the eye when the abdomen was opened.

We frequently noticed peristaltic constrictions of the antrum pyloricum when the rubber bag, of about 12 inches in diameter was at the cardia or fundus, and recorded no movement but that due to the pressure caused by the descent of the diaphragm. We concluded after 3 months experimentation that a small intra-gastric apparatus could not possibly record every peristaltic movement.

Sometimes one could witness very strong tonic contractions of seemingly every muscle fiber of the stomach;— it gave that impression, — by which the whole organ contracted from all sides by

shortening of every circular, oblique and longitudinal fiber, and at the same time the bag gave no record of movement, although when it was lying in the fundus it was clearly being lifted up — it would not record until it was compressed by food or the opposite gastric wall.

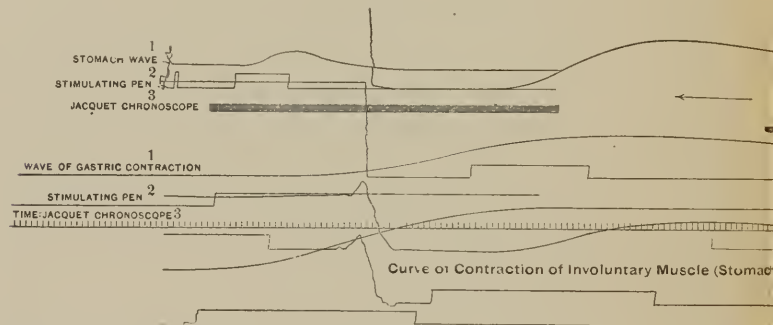
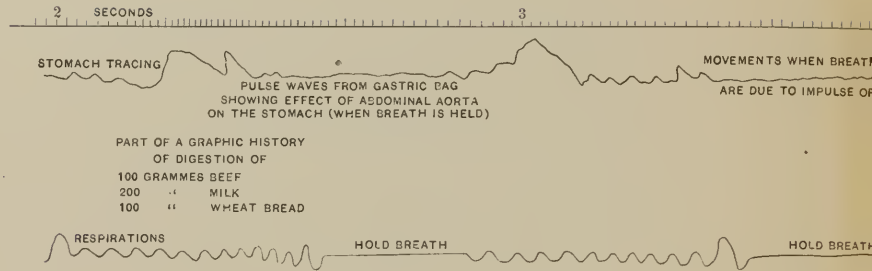
For these reasons, a bag was devised which had the exact shape of the stomach, but could readily be swallowed and when distended within the organ, exactly adapted itself to its interior filling every nook and corner in it. If a little food was needed in the organ we simply did not blow the bag up as far as to fill it out completely.

Our apparatus, as has been demonstrated to you many times on a large variety of cases in the clinical amphitheater is adjusted with great ease even in patients who are examined the first time. By a pneumograph it records the respiration separately and thus enables one to differentiate the active from the passive movements.

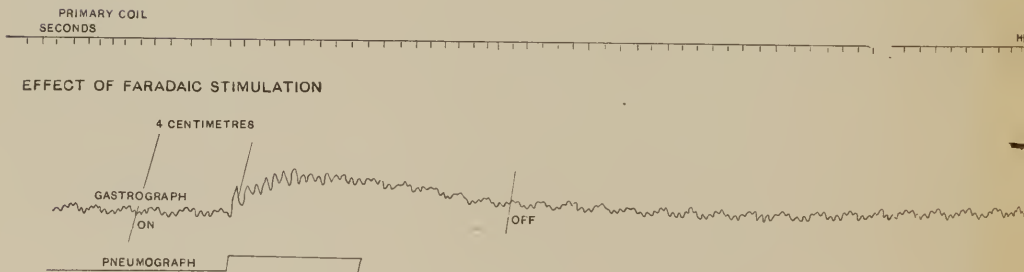
A separate seconds pen gives on the same paper a record in time so that the experimenter can at a glance tell the duration beginning and end of the peristalsis. Whilst it is the most perfect apparatus yet devised for recording the motor function it offers a reliable means of ascertaining the size and exact capacity, and finally the intra-gastric pressure. No apparatus hitherto devised combines these facilities in such a simple bit of mechanism for taking away the chymograph which should be in every medical school, its important parts are simply the intra-gastric bag and a manometer.

In practice a manometer connected with the intra-gastric bag will answer, with watch in hand the experimenter is able to count the peristaltic movements as they are conveyed to the column of water. Dr. Einhorn in his new book "Diseases of the Stomach page 96" has gathered the impression that the apparatus is of difficult adjustment because in my first report (l. c.) I stated that only such patients are taken as have become accustomed to the stomach tube as the nausea and vomiting first attending the initial introduction of the tube make an *exact* record impossible, I lay great stress here on the word *exact*, no intra-gastric instrument, not even Einhorn electrode can be introduced the first time without some nausea whilst this may not lead to emesis, it nevertheless has a great influence on the number of gastric movements as all cases we have tried generally show more contractions in the first experiment than in any

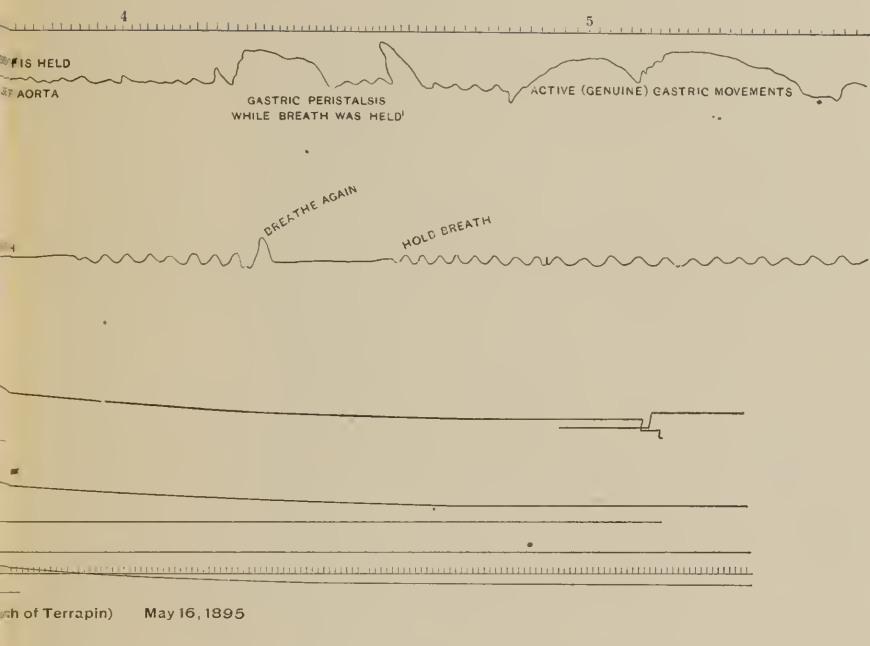
PLATE IV - V



Curve of contraction of stomach of terrapin, in which slow stimulations—
number of stimulations can be so increased per s

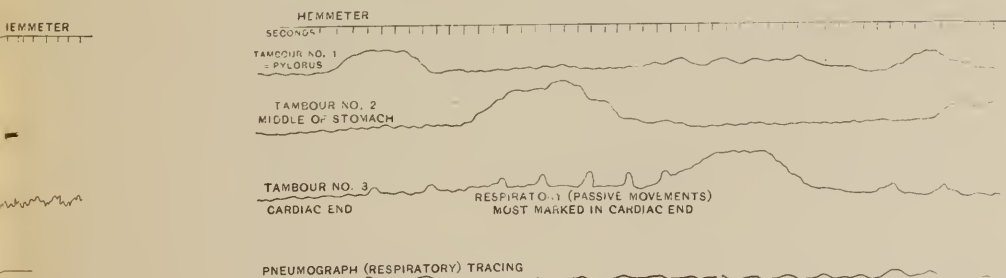


The showing of the cardiac impulse, as shown in the gastric record, is not a genuine cardiac inhibition, but only a
one, due to the fact that the stomach draws away from the diaphragm and aorta during violent contractions and does
every impulse; the radial pulse during this period was undisturbed and regular. Distance of secondary coil from primary
centimetres on the sliding apparatus.

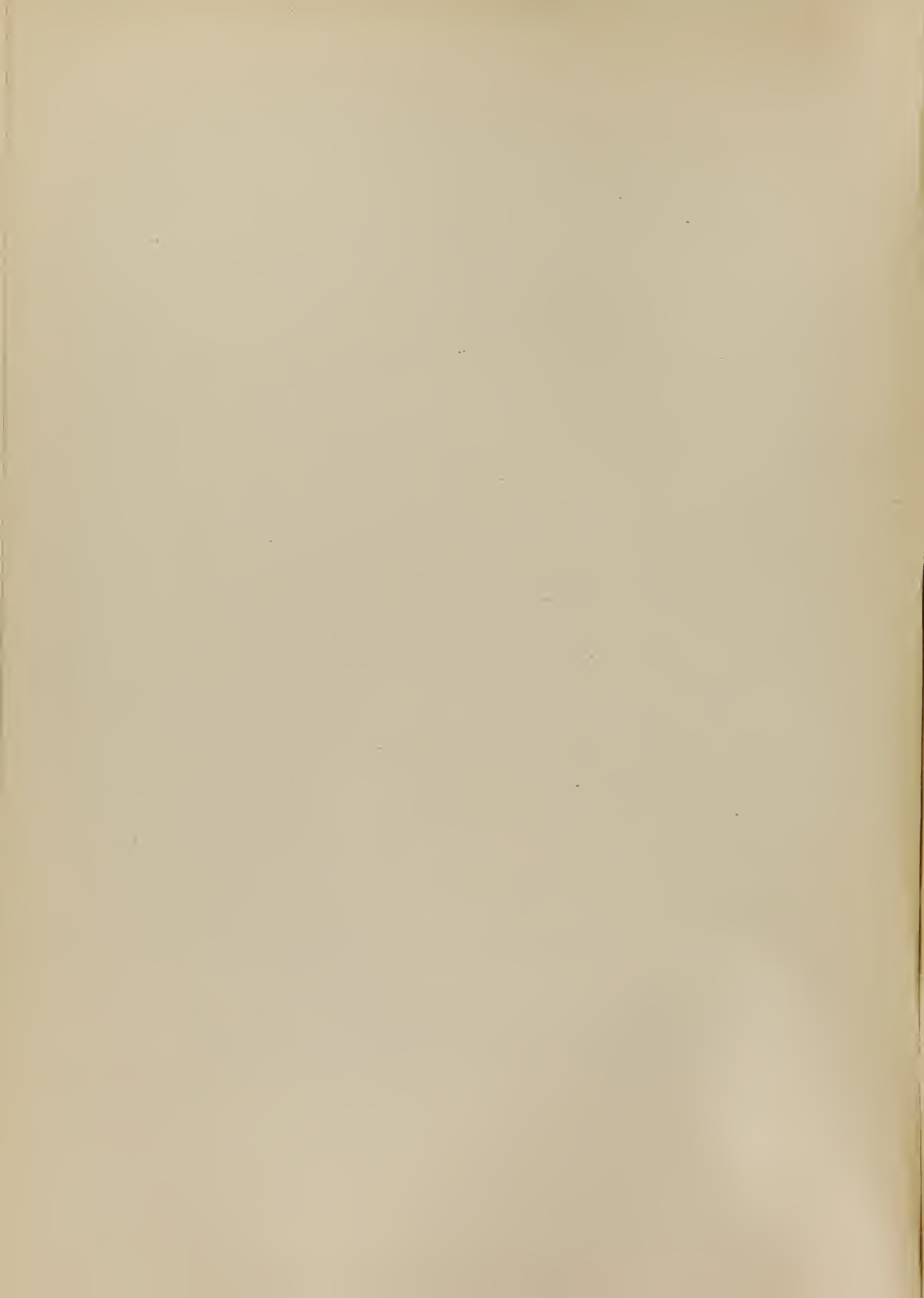


of Terrapin) May 16, 1895

i.e., twenty per second—are more effective than rapid stimulation. The second that the muscle will not contract at all.



Hemmeter's triple intragastric bag. Kymographic record of pyloric (No. 1,) middle portion (No. 2,) and caodiac end of stomach (No. 3,) in successive peristalsis.



other. If the record is to be exact and free from objections that may be urged on account of the influence of nervousness, nausea, suggestion, etc., a certain adaptation and experience of the patient is indispensable, no matter what instrument is used. None of these apparatuses will probably be regularly used in practice, they are implements for the trained specialists who know how to apply them and how to interpret their results. Nevertheless you have seen our intragastric bags used regularly at the Maryland General Hospital and good results obtained thereby, even at the first experiment.

My objections to the Einhorn gastrograph are that (1) no differentiation between active and passive movements is possible thereby, (2) that there is no coincident record of time in seconds on the paper, (3) that the tonicity or intensity of a contraction can not be adequately determined, (4) that the slow but very extensive general tonic contractions, a narrowing down as it were of the entire stomach to one point in the centre, will probably be recorded by a single dot such as would be made by an inspiration also. At the same time, when we reflect that a bag 12 inches in diameter may miss some of the contractions and fail to record them, it is difficult to imagine, that the gastrograph should record them all being not even an inch in diameter.

Nevertheless Dr. Einhorn's apparatus marks an epoch in the history of stomach motions and their physiology, it is the first attempt and largely a successful one to obtain their record by mechanical means. It seems of very easy application and when combined with an exact time record it permits of results that for practical purposes are sufficiently accurate.

Passive motions caused by the pulsations of the aorta and the impulse of the heart ventricles against that part of the saccus coecus cardiae which touches the arch of the diaphragm, and also the respiratory passive motions due mostly to the muscles of respiration are to a small extent participants in the causes of gastric movements, but they can not of themselves produce evacuations of the contents as we had occasion to observe in the clinic on the hysterical girl. R. H. who had no active stomach movements, no genuine peristalsis at all, all of her gastric movements were due to respiration and circulation.

This girl showed normal state of the secretions after an Ewald

test meal, but at the same time there was stagnation and over retention of food. It is therefore most essential to be able to distinguish between active and passive movements, for a person may have a great many movements of the stomach and yet have no genuine peristalsis at all.

LECTURE IX.

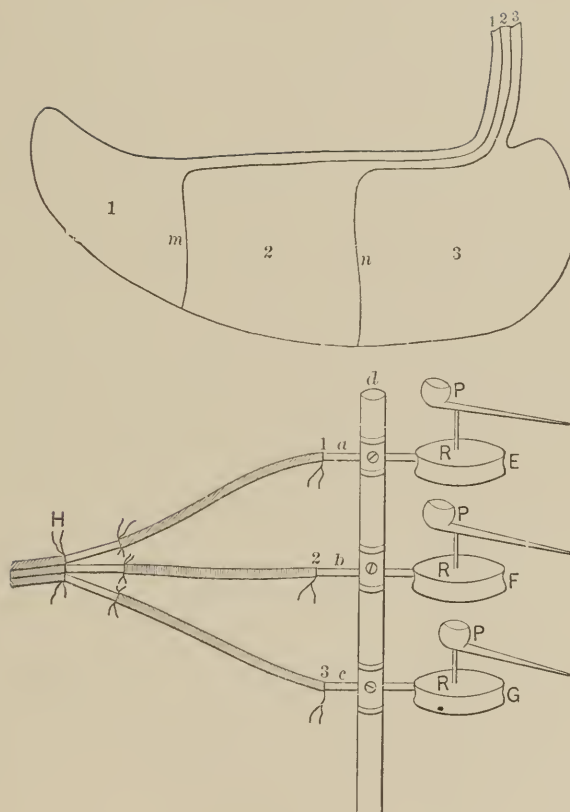
MOTOR FUNCTIONS OF THE STOMACH.

ONE OF THE intra-gastric, rubber, stomach shaped bags which are used in our clinic, consists of three separated compartments; one filling out the pylorus, the second filling out the middle portion of the stomach—the fundus, the third occupying a small part of the fundus and the saccus coecus cardiae. (See New York Med. Journal, Sat., June 22, 1895, page 772.) Each one of these compartments wrote its record on the kymograph by a separate tambour.

In that article I made the assertion from the results obtained with this bag that in the human being most if not all the peristaltic waves start at the pyloric end. This statement was made before Moritz's investigations in the *Zeitschrift für Biologie* were published, proving that the cardiac end and the fundus of the stomach could not contract, even when stimulated by powerful faradic currents on both, the mucous as well as peritoneal surface.

One week before my results were published in the New York Medical Journal, Dr. S. J. Meltzner of New York, published his results with direct and indirect faradization of the digestive canal, which demonstrated quite conclusively that the mucous membrane of the digestive canal offers a considerable resistance to the penetration of the faradic current to the muscular coat, the greatest resis-

PLATE VI



Intragastric tissue rubber bag, with three distinct parts and three separate outlets for recording the origin and direction of gastric peristalsis. Outside of the mouth the triple tube separates into its three component tubes, each being connected with a separate tambour and glass ink pen, writing the gastric contractions and relaxations on the kymograph. Part of bag No. 1 records the contractions of the pylorus; portion of No. 2, the middle of stomach, and part No. 3, of the cardiac end.

tance being found in the mucous membrane of the stomach. The percutaneous and the direct faradization of the stomach or intestines, can not produce any contraction in these parts.

Meltzer stated explicitly the kind of instruments used, the sliding inductorium (Schlitten apparat) of Du Bois Reymond — a Groves cell prepared anew for each experiment, and also stated the distance in every case of the primary from the secondary coil. His device of including the sciatic nerve of an animal (nerve muscle preparation of hind leg of frog most likely) in the circuit is most ingenious and practical.

There is however a very important matter which physiologists must insist on knowing, and which Dr. Meltzer does not state, perhaps because it was not very readily found out, and that is the number of stimulations to the second used by him. Involuntary muscle fibers are much slower to contract than voluntary muscles, and in electrical stimulation experiments, they contract much more readily when the number of stimulations does not exceed 240 per minute.

The vibrator on the Du Bois Reymond inductorium was found after months of experimentation to send too many stimulations into the stomach per second, when I later on used the Kronecker interrupter connection with a Jacquet chronograph and no more than 200-240 stimulations per minute, it was found that the pre antral sphincter could be made to contract with the distance of primary from the secondary coil = 0, and both electrodes on the mucosa.

To get this result, it is best to make the animal starve for 12 hours, for some reason yet unknown, the contractions are more unlikely to occur, the closer the experiment is made after the ingestion of food. Still it must be emphasized that practically the mucosa of the stomach is a nonconductor. I had occasion to try this in the physiological laboratory recently with a bit of healthy human stomach mucosa, that a member of this class tore off from the wall of his stomach during experimental lavage, the piece was 15 m. m. long, 5-6 m. m. broad, and 2-4 m. m. thick. The gentleman in question, after trying to wash his stomach out and not succeeding to his satisfaction, connected the end of the tube with a suction apparatus (aspirator).

This was followed by copious haematemesis for which I was hastily summoned. In the stomach tube, partly projecting from the

lower opening was a bit of fleshy substance which on microscopic examination proved to be gastric mucosa. After the hemorrhage ceased the young man was treated as if he had gastric ulcer for 1 week. He did not experience any pain during the accident nor thereafter, the only thing that frightened him was the blood. He made a good recovery. This bit of mucosa was placed in a continuous circuit with a milliamperemeter; the instrument showed 20 milliamperes with circuit uninterrupted, with the bit of gastric mucosa in the circuit it showed but 3 milliamperes. As it was impossible to get this fresh piece of mucosa into the circuit perfectly dry it is probable that the indication of 3 milliamperes was brought about through the conducting agency of the moisture on the outside of the tissue.

In the biological laboratory of the Johns Hopkins University, I have frequently had persons' stomachs connected with the kymograph, and an intra-gastric rubber bag blown up to fill out their stomachs exactly. Through the intra-gastric bag ran two insulated wires, one ending in a small brass knob near the pylorus, the other coming out against the mucosa in a similar knob near the cardia.

Every active and passive motion was recorded by a manometer pen. (N. Y. Med. Jour. June 22. '95 page 771.) But the strongest faradic currents (distance of primary from secondary coil = O) could produce no contractions of the stomach.

Dr. Geo. P. Dreyer and myself held one of the poles in our right hand, the plus for instance whilst the negative was in the stomach, with the left hand we touched the back of the person's neck, the current was so strong that it became intolerable to us, although this current made its circuit through the patient, it caused no contraction as evidenced by the manometer in connection with the intra-gastric bag.

Frequently we could observe contractions of any skeletal muscle upon which the outer electrode was placed—for instance the gastrocnemius and still the stomach did not contract, this proves that in some conditions the gastric muscosa may transmit a current yet the muscular layer give no evidence of contractions. I do not wish to imply that it is absolutely impossible to contract the human stomach by electrical stimulation, but the current required to effect this must be so strong that the experiment becomes hazardous.

Einhorn (Diseases of the Stomach, p. 78-83.) and Paul Cohnheim (Archiv f. Verdauungs krankheiten, Bd. I Seite 274.) have described tiny bits of mucosa which are found in the wash water and vomit of many gastric sufferers. I can confirm this observation and add that I have found these pieces of gastric mucosa on washing out the stomachs of perfectly healthy persons.

Now it has occured to me that rare instances in which a good contraction of the stomach was obtained, it was due to the fact that the current found its way to the muscular layer, through spots from which the glandular layer had been cast off. It must not be omitted to add that all stomachs experimented upon by my method in this series were washed out prior to the experiment to insure absence of current interrupting food particles in the organ.

Prof. Moritz of the University of Munich experimented with an apparatus very similar to mine, except that his rubber intragastric bag was round, not stomach shaped, it did not therefore exactly and completely fill out the organ, nor did he use the graduated pressure bottles by which it is possible to determine just exactly how much air is blown into the bag. Instead of a pneumograph, he used a cork in one nostril of the patient which was connected with a second manometer writing on the Ludwig kymograph; the advantage of the pneumograph over this method must be apparent to every one.

My first results appeared in print, three months before those of Moritz in the Zeitschrift für Biologie Bd. XXXII. Prof. Moritz has made the most important contributions to the physiology of the motor function since the investigations of Hofmeister and Schütz (Archiv f. exper. Pathol. und Pharm. 1886 Bd. XX.) In order that you may better understand the mechanism of the gastric peristalsis it is well to bear in mind the arrangement of the muscular layers, (1) longitudinal, (2) oblique and (3) circular, and what was said under the head of anatomy of the gastric layers and the formation of the sphincter of the pylorus. The stomach end near the pyloric part is spoken of more specifically as the antrum pylori.

The line of separation between the antrum pylori, and the body or fundus of the stomach, is made by a special thickening of the circular fibers forming what is spoken of as the transverse band by older writers, for instance Beaumont in his Physiology of Digestion 2nd. edit. 1847 page 104 — (a pioneer piece of work, very funda-

mental and thorough in its observations, this book remains a monument to American Physiological Investigation). Recent observers describe this transverse band as the sphincter antri pylorici and locate it at a distance of 7 — 10 centimetres from the pylorus.

In the antrum pylori, as you remember there is a very strong musculature and its glands contain only (or rather mostly) chief, central or ferment cells. The exact character of the gastric movements during digestion have been first carefully studied on the human being by Beaumont, his facts and errors have influenced physiologists more or less up to the present time. One can not fail to suspect that the stomach of Alexis St. Martin was too far from a normal one, to draw absolutely correct conclusions from. The extensive adhesions which Beaumont describes, certainly acted at times as irritants at others as impediments to normal peristalsis.

Prof. W. H. Howell's views on the gastric movements as expressed in his new American Text Book of Physiology, page 317, will serve as an expression of a modern specialist in this branch. He says (l.c.) the movements occur in two phrases: first, the feeble peristaltic movement running over the fundus chiefly on the side of the great curvature, and resulting in pushing the fundic contents into the antrum; secondly, the sharp contraction of the sphincter antri pylorici followed by a similar contraction of the entire musculature of the antrum, both circular and longitudinal, the effect of which is to squeeze some of the contents into the duodenum.

It is possible that either of these phases, with especially the first, might occur at times without the other, and in the first phase it is possible that the longitudinal fibers of the stomach also contract, shortening the organ in its long diameter and aiding the propulsive movement, but actual observation of this factor has not been successfully made. It can well be understood that a series of these movements occurring in short intervals would result in putting the entire semi-liquid contents of the stomach into constant circulation.

The precise direction of the current set up is not agreed upon, while it is probable that the graphic description given by Beaumont is substantially accurate. A portion of this description may be quoted as follows: The ordinary course and direction of the revolutions of food are, first after passing the oesophageal ring, from right to left, along the small arch; thence, through the large curvature from

left to right. The bolus, as it enters the cardia, turns to the left, passes the aperture, descends into the splenic extremity, and follows the great curvature into the pyloric end; it then returns in the course of the small curvature.

The average time taken for one of these complete revolutions, according to observations made by Beaumont seems to vary from 1 to 3 minutes.

It is possible, of course, that this typical circuit taken by food may often be varied more or less by different conditions, but the muscular movements observed from the outside would seem to be adapted to keeping up a general revolution of the kind described. The general result upon the food may be easily imagined. It becomes thoroughly mixed with the gastric juice and any liquid which may have been swallowed, and is gradually disintegrated, dissolved, and more, or less completely digested, so far as the proteid and albuminoid constituents are concerned.

The mixing actions are aided, moreover, by the movements of the diaphragm in respiration, since at each descent it presses upon the stomach. The powerful muscular contractions of the antrum, serve also to triturate the softened solid particles, and finally the whole mass is reduced to a liquid or semi-liquid condition in which it is known as chyme, and in this condition the rhythmic contraction of the muscles of the antrum eject it into the duodenum.

The rhythmic spiriting of the contents of the stomach into the duodenum, has been noticed by a number of observers through duodenal fistulas in dogs, established just beyond the pylorus. It has been shown also that when the food is entirely liquid — water for example — the stomach is emptied in a surprisingly short time, within twenty or thirty minutes; if however, the water is taken with solid food, then naturally the time it will remain in the stomach may be much lengthened.

Brinton [Diseases of the stomach] advanced the view which differs from Beaumont's in assuming a central current of the food, moving from the pylorus to the cardia through the central long axis in the stomach. There are according to this author two currents, one along each curvature running from the cardia to the pylorus, meeting and turning inward toward the centre of the stomach in front of the pylorus and then running back toward the oesophagus as a single

central current, there dividing to make again a current as before along each curvature.

According to Poensgen, (*Die Motor: Verricht: des Menschl: Magens-Strassburg* 82.) Reymond, Donders and Lesshaft approved of this theory, — whilst Penzoldt and Foster accept the great food circle of Beaumont.

Although I have made over 50 experiments on dogs, cats and rabbits to observe a food circulation within the stomach corresponding to these views, and although I have had an opportunity of seeing into the human stomach, through a fistula during digestion — I have not been able to confirm by actual observation either Beaumont's or Brinton's views. Whilst I have no new explanations to offer, it has occurred to me that the piston like, backward and forward movements of the food caused by the antral contractions, and especially of the sphincter of the antrum, is a sufficient force to effect the mixture of the chyme with HCL and the ferments, such as is found in it when it leaves through the pylorus.

The views of Beaumont and Brinton date from the epoch, when it was considered all important that food must be properly digested and macerated in the stomach, it was not conceivable then that by far the main bulk of digestion is carried on in the intestines. Hence the complicated theories of Beaumont and Brinton of circular movements of food owe their origin to the thought that such a movement was necessary to mix the ingesta with the gastric juice. In dogs this is not proven to occur in every instance; In herbivora (horse, cow.) the centre of the food mass in the stomach may be alkaline or neutral in animals killed one hour and a quarter after feeding.

In a number of experiments in which the stomachs of animals on opening the abdomen were found in active motion; I inserted long needles through the gastric walls to determine the direction they would assume under the pressure of the ingesta; according to Beaumont, the ingesta moving from the *saccus coecus* along the greater curvature to the pylorus, should compel the points of the needles to be directed toward the pylorus when run through the greater curvature, and at along the lesser curvature they should point toward the cardia.

If Brinton's theory were true, the points of the needles at both curvatures should at least during a large period of gastric digestion

be directed to the pyloric end. If needles are inserted to a distance of $\frac{1}{2}$ inch along both curvatures during active gastric peristalsis, a great diversity of movements of the outside portions of the needles is observable. They very rarely point the same way along either curvature, and one portion of them may point toward the cardia, whilst another points to the pylorus. Only when the needles are inserted very deep so that they dip into the central or axial stream, one can occasionally observe what appears as concerted action.

During active peristalsis, when the preantral sphincter at times contracts so powerfully as almost to obliterate the lumen, those needles inserted into the fundic portion of both the greater and lesser curvature are strongly turned toward the cardia, but simultaneously those few needles in the antral and pyloric portion are turned toward the duodenum. The same evidence of a central or axial current which indicates the pumping work of the muscular antrum in pushing back solid particles into the fundus, and squeezing liquid and semi-liquid portions into the duodenum, can be obtained by the intragastric electric lamp when introduced during the height of gastric digestion. These lamps can be seen through the abdominal wall in dogs whose abdomen has been shaven, when introduced in a dark room, though naturally not quite so distinct as when the abdomen is opened.

Once I observed this axial food current at the clinic in a female patient with very thin abdominal parietes, when the Einhorn intragastric lamp was introduced one hour after a meal. In animals with abdomen opened, I have been able to see this lamp carried along the entire greater or lesser curvature—during active digestion, but the occurrence is so rare as to appear accidental.

That the retrogressive current which is set up by contractions of the antrum, forcing the too solid food particles back toward the fundus, must inevitably set up some new movements among the remaining food mass in the fundic end is natural, but I doubt whether it ever reaches that systematic circulation described first by Beaumont and Brinton.

It should not be overlooked that if the observations of Beaumont of a complete food circuit were really true and the only movements in addition to the duodenal extrusion which the food mass underwent, there must always be a mass of food in the center of the

stomach which never touches the gastric wall if all the food moves about along the periphery, there must be a central quiet portion.

Brinton was aware of this defect in Beaumont's statements and improved upon them by his still more complicated piston movements to explain the axial food motions.

If the conditions described by these authors exist, they are not well explained by the arrangement of the muscularis of the fundus, which, as far as the work of Meltzer, (l.c.) Moritz (l.c.) and Goldschmidt (l.c.) show, is very feeble indeed in its contractions, and hardly sufficient to propel food in any direction, yet according to the above theory, much work is ascribed to it, as the preantral spincter is only 7 – 8 cm. from the pylorus it certainly cannot be made accountable for the movements all around the cardia and the saccus coecus.

The musculature of the fundic end has never been observed in peristaltic motions by myself except those that occasionally arising from the antrum travel upward over it. During active peristalsis it is in a condition of tonic contraction which with the intragastric bag in the fundus, I have estimated is equal to 6 – 8 centimeters of water. (Water manometer.)

Moritz, in his work on the motor function of the stomach, studiously avoids referring to any systematic food circulation within the organ. It seems rational that sufficient churning and mixing is effected by the powerful contractions of the antrum during the general tonus of the fundus to explain the saturation and softening of the ingesta by gastric juice.

The contrasting relation of the fundus and antrum regarding active peristalsis, are evident in the degree of pressure, as observed on a water manometer in connection with my triple intragastric bag. In the fundus, the pressure is on an average equal to 3 – 6 cm. of water. The increase of intragastric pressure due to cardiac action is equal to 1 – 2 cm. (In this is included the pressure due to every new heart impulse and aortic impulse.) The inspiratory increase of pressure is equal to 6 – 12 cm. These are very nearly the figures Prof. Moritz obtained before me, and, I add them here merely in support and confirmatory of his views.

The physiology of the motor function has been dwelt upon more extensively than seems necessary in a condensed statement of

gastric pathology, not only because it is the most important office of the stomach, but because I have become convinced that in a large majority of disorders of secretion and absorption, (not all) an abnormality in the motor function lies at the foundation.

The exaggerated or diminished peristalsis can on careful examination be detected sometimes before the secretory and absorptive anomalies are apparent. The secretory disturbances observed after double vagotomy (section of both vagi) are due according to Contejean to the motor paralysis caused at the same time. (*Archiv de Physiologie* 5, IV, p. 640.) A similar view is held by H. Borutteau—(*Phlügers Archiv*, Bd. 65 p. 26.)

The relation between motility and secretion and absorption is not at all well understood, the peristaltic movements effecting a churning motion are those mostly concerned in stimulating secretion, when these movements are lost, secretion is generally lost also.

The last vestige of peristalsis left, is that by which the stomach is emptied, and it may be present with total absence of secretion. In stomachs with motility much impaired and secretion arrested the absorptive function is greatly reduced—(Atrophic Gastritis Carcinoma.) In temporary arrest of these functions, the secretive and absorptive functions generally return with improved motility.

LECTURE X.

ABSORPTION FROM THE STOMACH — PENZOLDT'S AND FABER'S, HERSCHEL'S, JULIUS MILLER'S AND HEMMETER'S TESTS FOR GASTRIC RESORPTION.

THE METHOD most commonly employed is that of Penzoldt and Faber, consisting of 3-5 grains of iodide of potassium enclosed in a gelatin capsule, which is administered with 100 c.c. = 3½ ounces of water. Iodide of Sodium or Potassium when taken internally, will appear and can be tested for in the saliva and urine where it is excreted in about 6½ to 15 minutes.

The test is made generally by wetting starch paper with saliva of the patient every 2 minutes after the KI is taken, and touching the wet spot with fuming Nitric acid. The first appearance of a blue color indicates that the iodide has reached the point of excretion in the saliva, and consequently must have been absorbed. If this reaction occurs later than after 15 minutes, then the rate of absorption is reduced, this according to Zweifel (*Resorpt. Verhältnisse d. Menschl. Magen* Deutch. Arch. f. Klin. Med., Leipzig, Bd. XXXIX, p. 349, 1886.) occurs in Gastritis, Dilatation and Carcinoma; in gastric ulcer, the resorption is said to be normal or nearly so.

Most authorities (J. Wolff, Zweifel, Sticker, Quetsch) differ very much on this matter, but agree on the reduced absorption in

carcinoma. If the iodide is given during a meal the reaction occurs much later.

Herschel — (Indigestion London, 1895, p. 115.) estimates the absorptive power by giving two decigrams of powdered rhubarb. This will give a red color in the urine with liquor potassae normally in 15 minutes. My experience with this method is that frequently the urine is so highly colored in digestive patients that the red color must be very decided to be recognized. In addition to which it suffers from the same objection as Penzoldt's and Faber's method. These are; — In the first place Brandl's experiments have shown that sodium iodide is absorbed to a very slight degree or not at all in dilute solutions.

Not until its solutions reach a concentration of three per cent or more does its absorption become important. Accordingly all soluble inorganic salts are practically not absorbed in the stomach, since it cannot be supposed that they are normally swallowed in solutions so concentrated as three per cent. Brandl also found that condiments, such as mustard, pepper, also alcohol, very much facilitated the absorption of sodium iodide, perhaps these substances act by stimulating the epithelial cells, or by causing a marked hyperaemia of the mucosa.

The absorption time does not vary much in the same individual, except, when the stomach is full; in this case it is not only prolonged, but is very variable in the same individual. This prolongation according to Sidney Martin (Diseases of the Stomach London 1895) is probably due to a considerable dilution of the iodide by the stomach contents, and also to the fact that the salivary glands are not so active some time after a meal as in the fasting condition. One must not overlook the fact in these experiments, that it is not only the absorptive activity of the stomach that is being investigated, but also the excretory activity of the salivary glands.

In Zweifel's experiments it is probable from what we know of the absorption of water in the stomach through the observations of Tappeiner (über Resorption im Magen—Zeitschr. f. Biol.—München Band XVI, p. 497, 1881.) and Von Mering (l.c.)—that most of the liquid containing the iodide passes rapidly into the duodenum. Therefore we may be testing not only gastric absorption and excretory activity of salivary glands, but also intestinal absorption.

Zweifel concludes (l.c.) that in all diseases of the stomach, there is a prolongation of absorption time which is greatest in dilatation and carcinoma and least in chronic gastric catarrh and very slight in ulcer in the later stages, in the early stages of ulcer however he claims absorption is also prolonged.

It is very evident that no differentiation between catarrh and ulcer is possible according to this method and thereby one of the main purposes of such investigations, that of aiding in the establishment of a diagnosis is thwarted.

In view to these defects which apply equally well to Herschel's, Penzoldt's and Faber's methods of testing absorption, and are caused mainly by the fact that water is not absorbed from the stomach, and that the varying secretory activity of the salivary glands is a factor influencing absorption time, I have devised a method which is available for experiments on gastric absorption in the physiological laboratory, and, which I have successfully tried on six male patients and ten healthy students. The manner of testing the urine or saliva was discarded entirely.

My method consists in washing out the stomach thoroughly, then, by means of my method of duodenal intubation, the entrance into the duodenum was plugged or closed up by introducing a small, rubber balloon into it and blowing it up just in front or beyond the pylorus. (This method has been described also by Dr. F. Kuhn in the *Münchener Medizin. Wochschr.* No. 27, 28 and 29, 1896, but his method is built upon a different principle from mine—the spiral electrode.)

After thus mechanically closing the pylorus, a weighed amount of any harmless inorganic salt, sodium chloride, or sodium sulphate dissolved in 100 c.c. of distilled water so as to make a 3 percent solution, is poured into the organ through a tube, this is indispensable to exclude loss of the salt solution through clinging to the tongue, mouth and oesophagus, or absorption from these tissues.

After a lapse of 10 minutes, the fluid is again drawn out of the stomach by aspiration, or even if necessary, by adding known quantities of distilled water until the last washing gives no indication of containing any trace of the salt by a proper chemical test. (In case NaCl sodium chloride or common cooking salt has been used, a weak solution of nitrate of silver AgNO_3 can be employed to assure

oneself that this last wash-water contains no more NaCl. The normal HCL is not secreted so rapidly as to cause confusion in the result.) This entire water is now evaporated to dryness and the residue weighed. The difference between the amount of NaCl poured into the stomach—which in a 3% solution is 3 grammes in case 100 c.cm. are used, and the amount regained indicates the degree of gastric absorption.

To simplify matters, the practical suggestion of Julius Miller (Boas' Archiv f. Verdauungs Krankh. Band I, p. 237.—Zur. Kennt. d. Sek. u. Resorpt. im Menschl. Magen.) has been utilized and can be recommended. It consists in noting the specific gravity of salt solutions before pouring them through the tube, and, after any desired time, the solutions are again washed out or aspirated and the specific gravity again determined.

The difference between these specific gravities taken before the salt solution enters the stomach, and, after it is regained, affords a satisfactory index of the rate of absorption from the stomach if escape of the solution into the duodenum is prevented. It is not necessary to evaporate the whole solution to dryness in case sodium chloride or any other harmless neutral salt is used. But after measuring the total quantity of liquid regained—say for instance it amounts to 1 liter = 1,000 c.c.—the amount of NaCl in 10 c.c. can be determined by evaporation in platinum, and, weight of the total remaining NaCl calculated by multiplying the result by 100 or whatever the figure may happen to be.

This method of determining the rate of gastric absorption gives approximately accurate results even without duodenal intubation and mechanical closing of the pylorus, provided that by several preliminary experiments the motility of the patient's stomach has been relatively determined.

By observing how much of 500 c.c. of water he will pass into the duodenum in—say 10 to 20 minutes, this also requiring the drawing out again of what is left of the 500 c.c. of water that were drunk. Von Mering (l.c.) found that of 500 c.c. of water given through the mouth of a large dog, within 25 minutes the entire amount, or at least 495 c.c. had been passed out of the stomach through a duodenal fistula.

In the human being the passage of water out of the stomach is

not near so rapid. Julius Miller (l.c.) found that the human stomach was not even rid of 200 c.c. NaCl solution of the specific gravity 1028 in 30 minutes — after this time he regained in one case 75 c.c., sometimes he regained more liquid than he poured in.

In 30 tabulated measurements which he gives with sodium chloride solution, (P. 240 l.c.) he regained more than he poured in—4 times, the same amount—once and a less quantity—25 times. But his figures go to prove that even with open passage into the duodenum, comparatively small amounts of salt solutions are passed out in 15 minutes.

Hence if in any individual the average amount passes into the duodenum in 15 minutes is known by previous experiments. Closing of the pylorus is not necessary to reach an approximate result concerning the rate of absorption. Miller confirms Von Mehring's conclusions that contemporaneous with absorption a secretion of water occurs into the stomach.

This secretion increases with the concentration of the solutions. In the four instances mentioned where more was regained than poured in, the specific gravities which are a good indication of concentration were 1066—1061, 1052—1088 and 1035. (Regarding the taste of 3% solution of NaCl, it might be explained that this is the percentage of the salt in the water of the Atlantic; Ocean water has been recommended for internal use—A. Levertin—Hygieina, XLVII 8 Svenska läkaresällsk Förh S. 138, 1885.)

In my studies with occlusion of the pylorus I experimented also with known solutions of sodium sulphate pepton, maltose, cane-sugar, milk-sugar and alcohol. As water is poured out on the surface of the mucosa, in return for salts absorbed, the S. G. will not always instruct us as to the contents of Na Cl — which had best be arrived at by weighing.

From experiments on animals, it is known that a concentrated solution may cause the stomach to secrete water, thereby diluting it, but that at the same time it is possible that there may be no re-sorption. So that weighing the residue from evaporating the liquid regained may be unavoidable for a correct result.

Maltose, I have found a very practical substance for absorption experiments, though dextrose will also answer this purpose. As their quantity can be readily determined in solution by titration with

Fehling's solution, and also by the fermentation test for which the Einhorn Saccharimeter is most serviceable. Maltose will not reduce as much Fehling's solution as dextrose, the exact relation between the two being according to Brown and Heron for maltose 60,8: dextrose 100.

According to Soxhlet 1 c.cm. Fehling's solution corresponds to 7.78 mgr. Maltose in 1% solution (Provided the Fehling's test was not diluted.) Though maltose is converted into dextrose in the stomach, the amount converted in 10 — 15 minutes is according to my observations small enough to be disregarded. If desired a test by Barfoed's reagent may be made to detect if any dextrose is present in the liquid regained.

The amount of sodium chloride in the solution regained can also be determined by titration. (Salkowski u. Leube—*Die Lehre vom Harn*, also Neubauer and Vogel—*Analysen I. Urins.*) The method is given in the Laboratory Manual of my assistant Dr. Edward L. Whitney. (An introduction into the Laboratory Methods of Clinical Pathology, page 18, Baltimore 1896.)—My method for absorption testing is:—

- 1 To determine the amount of 500 c.c. of a 3% NaCl solution passed into the duodenum in 10 minutes.
- 2 Allow 500 c.c. 3% NaCl solution to run into a clean stomach through a tube and remain 10 minutes.
- 3 Draw out as much as possible, washing out the last with known quantities of distilled water.
- 4 Determine the amount of NaCl as stated above, and add the average deficit of escape into the duodenum.

The difference between the original amount NaCl and the amount regained is a fairly accurate index of gastric absorptive power, or, by my method of duodenal intubation, occlude the pylorus by blowing up a balloon in front or beyond it;—Pour into the stomach through a tube a known quantity—say 100 c.c. of a 1% of maltose;—In 10 – 20 minutes, aspirate or wash out the amount of maltose left as above, the deficit will invocate the amount absorbed.

LECTURE XI.

METHODS FOR DETERMINING THE LOCATION, SIZE AND CAPACITY OF THE STOMACH — GASTRO- DIAPHANY OF EINHORN.

THE CONVICTION has been forced upon me that the degree to which the stomach can be distended is a very limited one.

This statement is made after many distensions with the intra-gastric stomach-shaped bag. Most stomachs that are in a normal state will refuse to be distended more than 100 c.c. beyond their natural capacity. Only in pathological thinning of the gastric walls and in atrophy of the muscularis is an over distension conceivable, even then some of the gases will escape by the cardia before painful distension will ensue.

For these reasons it is that distension with air or carbon dioxide is an expedient and safe way of determining the form and location of the stomach, and, its relation to any \times tumors that may be present. Riegel thinks that there is no better way of differentiating gastric dilatation from gastropsis (falling) than by this process of distension.

This method is carried out by introducing a stomach tube, to the upper end of which is attached a double bulb, pump arrange-

ment — such as is used in some spray apparatus. (Runeberg—Deutsch. Archiv f. Klin. Med., Bd. XXXIV.) — Bouveret (Traite des Maladies de l'estomac, Paris, 1893.) recommends that the air be blown into the stomach, by blowing with the mouth through the tube. Riegel and Boas are very fond of gastric distension by carbon dioxide gas, which is done by filling two tumblers, each half full of water, in one of which we usually dissolve a teaspoonfull of bicarbonate of sodium, and in the other about the same amount, perhaps a little less, of tartaric acid.

First the solution of tartaric acid is administered, and immediately afterward the sodium bicarbonate; within the stomach a brisk evolution of CO_2 occurs at once distending the organ, whereby it stands out prominently and is evident as a sharply defined arched elevation, the greater curvature becomes very apparent, not so the lesser one.

The stomach under distension can be readily palpated or percussed. If tumors were made out before the distension, it is important to determine their seat after the distension. It is possible in many cases to demonstrate the connection or non connection of the tumor with the stomach after distension. The movability of the tumor gives us some information as regards its seat, particularly if it be carried out before and after distension, for under this method the organ is not only stretched out more, but undergoes a certain amount of turning, or twisting around its long axis.

Accordingly tumors, which, when the stomach was empty, were palpated in the line of the umbilicus and to the right, and for that reason might be doubted to belong to the stomach,—after distension — may move upward to the right, and toward the anterior arch of the short ribs. One may see, and feel the direct transition of the tumor mass into the substance of the stomach, or to trace its extent over the small curvature toward the pylorus, or ascertain that it is entirely independent of the stomach.

Even the disappearance or the becoming less distinct of a tumor, is very important if it occurs after distension. This is observed in tumors of the posterior wall. If it is easily movable, very close and tight adhesions may be excluded, if it is absolutely immovable it is abnormally attached or fixed. So you will recognize that distension of the stomach with air or gas does not only enable one to

get a better percussion area, but it serves another purpose that of facilitating the palpation of tumors. In his new book on Diseases of the Stomach, Riegel gives in addition to the above, ten other methods.

LOCATION, SIZE AND CAPACITY.

For determining location, size and capacity of the stomach, all of which are more or less fallacious, and I must refer those specially interested in this matter to Riegel's book, p. 41-56. In my opinion, all of these methods will, before many years, have only a historical value. There is one method for accomplishing above objects, however which I can recommend from a very large experience with it, and, which is used exclusively at my clinic and with the accuracy of which you have had many opportunities to be convinced. It is described and pictured on page 32 of this work.

With my stomach shaped, intragastric, rubber bag (see p. 32.) and the pressure bottles A and B, the location and capacity can be determined with great ease. The rubber bag used for this purpose has no sheath or guide for the duodenal tube. The stomach is distended by blowing up the bag within it, the amount of air necessary thereto is measured afterwards by allowing it to escape into a spirometer; a less accurate, though a quite practical method is to catch the escaping air in a glass cylinder filled with water and inverted over a basin.

It might be claimed that my method is a combination of von Kelling's, Schreiber's and Jaworski's methods, and, it does indeed partake of part of the devices of all these three. (See Riegel, pages 51, 52 and 54.) Schreiber used a small, round—not a stomach shaped—distensible balloon, but no pressure bottles or spirometer. Jaworski used two pressure bottles, but no balloon or intragastric bag and no spirometer, whilst von Kelling used simply the spirometer to measure the air which he forced into the stomach with a double bulb as is used on sprays.

My method of arriving at the capacity of the stomach is really then not entirely original, as it combines the best of three older methods, but it is most convenient and reliable, the bag, as has been shown to you, can at the same time be used for determining the nature of the motor function. It can be asserted from observations

on a large number of patients, that there is no single method in existence which is so useful and can combine an instruction concerning size, location and capacity of the stomach with that concerning its motor function.

The method is as easy in its application as any which Riegel describes. The capacity can for practical purposes be read off on bottle B, from the amount of air that has been displaced into the intragastric bag. The expense of the bag is \$1.00, and good results of the motor function can be gotten from a water manometer in connection with it after distension; with one hand on the epigastric region, the respiratory movements can be felt and thus distinguished from the active movements as expressed by the rise and fall of the water column in the manometer. It may thus be used without the kymograph.

In the shops of Baltimore, small toy balloons are sold; they are made of very thin but quite tough rubber, which, my assistants have frequently used for intragastric distension. These balloons accompany a game called "pillow dex" and are sold 6 for 25 cents. For studying the motor function they answer as well as the expensive stomach shaped bags, as I have assured myself that on distension they fill every inch of space in a dog's stomach. — For determining the capacity however, the stomach shaped bag is more accurate.

GASTRO DIAPHANY OF EINHORN.

In 1889, my friend Dr. Max Einhorn, succeeded in transilluminating the human stomach in the dark by means of a small Edison lamp attached to a soft, rubber tube; from the lamp through this tube, insulated conducting wires run to a storage battery. (See illustration.) At some distance from the rubber tube is a current interrupter. By this apparatus the inventor claims to be able to ascertain the exact position and size of the stomach and to recognize tumors and thickenings of the front wall by their lack of translucency.

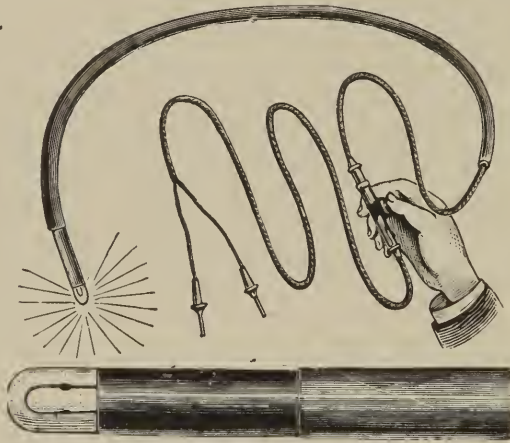
In 1867, Milliot had succeeded in trans-illuminating the stomachs' of animals by platinum wires contained in glass tubes and connected with a Middeldorph's apparatus.

Fleischer—in his Text Book—(Path. u. Therap. der Magen. u.

Darm. Krankh., p. 789.) — claims to have succeeded in transilluminating the human stomach together with Hüfler before Einhorn. If this is really so, Fleischer did not publish his investigations as far as I know, and, certainly is not entitled to call the method after himself—The Gastro Diaphany of Fleischer and Hüfler.

To Einhorn is due the credit of presenting the method as an aid to Diagnosis. You have all witnessed the application of this method in my clinic;—The patient in a fasting condition, drinks a liter of water, the apparatus is passed into the stomach just as the lavage tube is passed and connected with the storage battery. The

Fig. 5.



THE ELECTRO DIAPHANE.

stomach transmits the electric light through the abdominal walls, becoming visible as a red zone at the place which corresponds to its location.

In case the anterior gastric wall is occupied by a tumor, the light will not be transmitted at that spot, but all around it the rays will penetrate, thus evincing a dark shaded area in a luminous zone.

As you know, I am in the habit of marking the ribs, particularly the umbilicus xyphoid cartilage, and symphysis pubes with phosphorus, so that they can be seen in the dark, and serve as land

marks to the exact abdominal area in which the light permeates. Prof. Howard A. Kelly from whom I received much kind advice, encouraged me to attempt transillumination of the colon by this method, and we succeeded admirably.

I have been able to illuminate in successive portions, the entire colon in this manner and demonstrated prolapse of the colon thereby, even into the duodenum have I introduced the diaphane, and claim these advances of the method as original to our clinic.

Notwithstanding the conservatism of Riegel and Fleiner, (Lehrbuch d. Krankh. d. Verdauungs Organe, page 223.) and the objections of Boas, I consider the method valuable—it certainly is convenient for the rapid diagnosis and the differentiation between gastrectasin and gastroptosis.

It should be added however that I use a much stronger light, (namely eight – ten volts) than Einhorn, and have one half of the lamp coated by a reflecting mirror of mercury, which can of course be controlled by turning the tube outside of the mouth. At a demonstration which I was requested to give before the Clinical Society of Maryland, the apex impulse of the heart was visible in the dark after transillumination.

LECTURE XII.

THE STOMACH TUBE AND TECHNICS OF ITS INTRODUCTION — EXAMINATION OF STOMACH CONTENTS — TEST MEALS; THEIR EFFECT UPON THE AMOUNT OF ACID SECRETED.

NEVER USE ANY other kind but a soft elastic stomach tube and before introducing it for the first time in any patient, always instruct him or her carefully about the object, and utility of the procedure and its harmlessness. Whenever I can do so I give very timid patients an opportunity of observing with what ease more experienced patients introduce the tube on themselves, this has a most comforting effect. Weak and old persons should always be treated on the bed — several thick towels are placed on the patient's chest and beneath the chin. I always use a linen gown if the case is to be examined in an erect position and over the breast and lap an additional rubber sheet. If the throat and fauces are very tender, (often found in excessive smokers) it is advisable to precede the introduction of the tube by spraying the throat with a 3% solution of Cocaine hydrochlorate or the following anodyne spray:—

R

3% solution of Cocaine hydrochlorate in Benzoinol	fl ʒi
1% solution of Menthol in liquid Vaseline oil	fl ʒss

Use in atomizer for spraying the throat.

It is not necessary to lubricate the stomach tube with any oil or vaseline, there is generally mucous enough in the oesophagus to facilitate the passage.

In the N. Y. Medical Journal for Dec. 28, 1895, Vol. LXII, No. 26, page 822, a new double current stomach tube has been described by the author, through which the inflow and outflow goes on uninterruptedly at the same time. This tube is recommended only as a time saver for the specialist in practice, the simple tube will fulfill every requirement, even though lavage of progressed gastrectasia may require much more time.

From twenty measurements of living female patients, the author

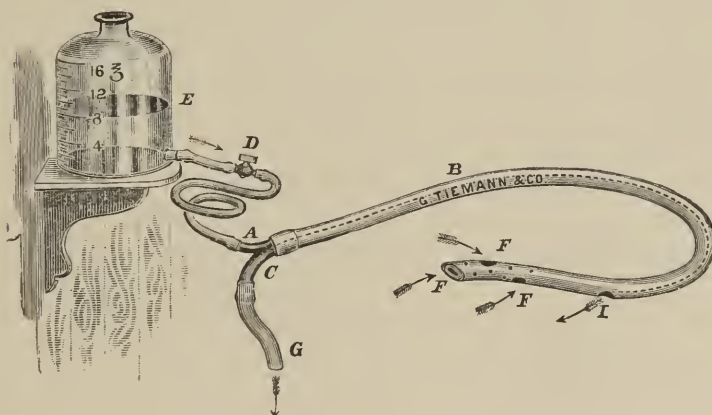


Fig. 6. Hemmeter's Double Current Stomach Lavage Tube.

has found that the average distance from the incisor teeth to the deepest portion of the stomach is fifty-five centimeters, and in thirty-six measurements of healthy males the same distance was found to be sixty centimeters. In cadavers this is in both sexes, according to the author's experience, shortened by post-mortem rigor, it having been found to be 52.5 centimeters for females in twelve different subjects on the average. In twelve male cadavers, the average distance from the incisor teeth to the deepest part of the stomach, was fifty-four centimeters.

In ten cases of dilatation of the stomach, the average distance from the incisor teeth to the deepest portion of the stomach, as

measured by as rigid a sound as could safely be introduced, was sixty-nine centimeters. In ordering the new double-current tube, however, from Tiemann & Co. the manufacturers were directed to make the portion which is introduced into the body, seventy to seventy-two centimeters long, which length, on the basis of the foregoing measurements, was considered sufficient for all requirements. The amount flowing down through the inflow tube, will vary with the

height of the pressure bottle, but should never exceed more than one litre in two minutes; in the same tube the outflow channel should be able at the same time to discharge by simple siphonage two litres.

The inflow and outflow tube should be tested as regards their calibre, the former by pressure, the latter by siphonage, before they are used on patients.

When the outflow tube is tested as regards the amount of water, it will discharge in a given time the tube should be arranged as in Fig. 7, so that the water must rise seventy centimeters, the distance from the deepest portion of the stomach to the incisor teeth, before it can descend into the measuring graduate,

The procedure of lavage must be attentively watched in cases where much solid *debris* is expected, and if the outflow becomes choked with solid matter, the inflow must be cut off at once, until the outward passage is made clear.

A piece of glass tubing two inches long, firmly tied to the outflow tube, is useful, in that it permits the operator to observe the material which is running out. The single stomach tube has been swallowed entirely and disappeared into the stomach. At least two such accidents are on record, one by Leube (33); the other by Jask-

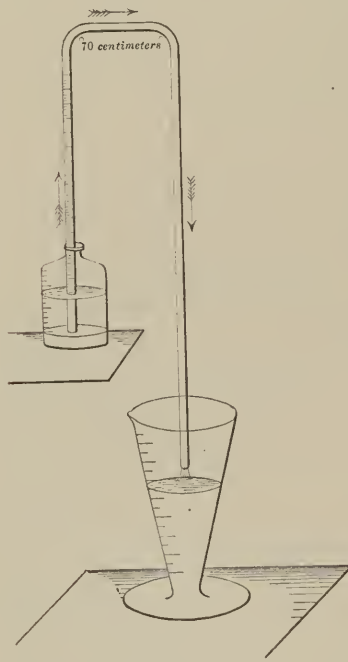


Fig. 7.

Illustrating the Principle of Siphonage.

son (35); they are both quoted by William H. Welch, in his article on Dilatation of the Stomach in Pepper's (American System of Medicine, Vol. II.

With a double or current tube, this accident is impossible, as the instrument is tied to the supply reservoir. Aspiration of the mucosa and tearing of pieces of healthy membrane by suction, which has occurred in Leube's experience (2), is also a matter of impossibility with the recurrent tube. Physicians who have used gastric lavage with a simple funnel, know how difficult it is to keep air from being sucked into the stomach; a deep depression of air forms in the centre and is occasionally aspirated into the stomach; all of this is avoided in the recurrent tube. The author on visiting Professor F. Penzoldt in Erlangen, in July, 1895, was surprised to find this pioneer of digestive pathology still advocating the use of a guide in the shape of a flexible stick or whalebone, which during introduction, is inserted into the gastric tube to facilitate its entering the œsophagus after it curves over the base of the tongue.

In his most recent contribution to the subject, Penzoldt (loc. cit., 27) gives minute details as regards the method of application of the *Leitungsstab* or *mandrin* within the tube, and says that it should be oiled to facilitate its removal when the tube has reached the middle of the œsophagus. He also suggests catching the tip of the lavage tube between the index and middle finger of the left hand, which are inserted into the patient's mouth, and bending the tip down over the base of the tongue until it enters the œsophagus. This is the method advocated by his teacher, Prof. Leube (loc. cit., 2), and also by Rosenheim (36).

In the writer's experience the intratubal whalebone guide and the insertion of the fingers into the patient's mouth are superfluous. The tube can always be introduced without a guide, and without touching the patient. The main point is that the point of the tube, when it has reached the wall of the pharynx, shall be deflected downward. This will occur without exception, and in a very natural, easy, manner if the patient is directed to swallow at this moment. In the moment of this act of deglutition the point of the tube is bent downward into the œsophagus.

Beginners in using the tube need have no fear that it will enter the trachea. To make it enter the trachea, is in the writer's exper-

ience, a difficult undertaking, and requires special training and dexterity. He was present on an occasion when a class of ten students were taking a private course in diseases of the throat, during which lesson they were trying to mop the larynx. What they really did was to mop out the superior portion of the œsophagus. Direct the patient to keep taking deep inspirations, and as soon as the tip or point of the tube is felt touching the pharyngeal wall, tell him to swallow, and almost immediately the tube follows into the œsophagus and can be pushed into the stomach without further resistance.

It is not necessary for the patient to open his teeth any wider than just to admit the sound; at the same time caution him not to bite on it, but to breathe naturally. No patient should be subjected to gastric lavage without previously examining the thorax. Penzoldt tells of a case in which the stomach should have been washed out in the morning, but on account of lack of time this was postponed until the same evening. On the same afternoon the patient died of rupture of an aortic aneurism into the œsophagus. This leads to the conclusion of this paper with a brief statement of the indications and contraindications of gastric lavage.

The recurrent stomach tube is not available in the removal of test meals from the stomach, because it needs too much water to operate satisfactorily, which would later complicate the analysis. If, however, this surplus water is no objection to the chemist, the double tube is a rapid means of obtaining gastric contents. The same indications and contraindications that hold good in applying the single tube appertain also to the double tube. They are these: the tube is contraindicated—

1. In all constitutional and local diseases which could be aggravated or life endangered by the irritation and exertion of lavage. Among these could be mentioned:—

1. Pregnancy (though this is not a disease).
2. Heart disease in a state of defective compensation—heart neuroses, angina pectoris, myocarditis, and fatty heart in advanced stage.
3. Aneurism of the large arteries.
4. Recent hæmorrhages of all kinds, including apoplexies, pulmonary, renal, vesical, gastric, rectal hæmorrhage and hæmorrhagic

infarctions.

5. Advanced pulmonary tuberculosis.
6. Advanced pulmonary emphysema with bronchitis.
7. Apoplexia and cerebral hyperæmia.
8. Advanced cachexia.
9. Presence of continued or remittent fever.

The stomach and intestinal diseases which Boas states are contraindications of the use of tube are:

1. Ulcer with recent hæmatemesis and dark stools.
2. Palpable carcinoma of the pylorus, with vomiting of coffee-ground material and the classical symptoms of cancer.
3. Many gastric neuroses in which the character of the malady is clear without lavage.
4. Stomach or intestinal troubles with acute fever.
5. Gastric mucosa easily started to bleeding.
6. Secondary gastric affections whose dependence upon a distinct and more important primary disease is evident.

These are not invariable rules, however; there may occur cases under some of these exceptions at times that on account of depressing self-intoxication from the stomach or advanced gastric fermentation peremptorily require lavage. Thus, according to Boas, it has been employed with success in pregnancy, and the author has once washed out the stomach in case of typhoid fever with favorable result, and also performed lavage in a case of aortic regurgitation with Bright's disease and gastrectasia where much relief was experienced from the procedure. Professor Moritz, of Munich, has frequently passed the stomach tube in pregnant women to ascertain the intra-gastric pressure (25).

In a normal position of the abdominal viscera the location of the cardia corresponds to the spinous process of ninth thoracic vertebra. By counting off this process on the back of the patient and placing the upper eye of the sound against it, one can measure the length of tube necessary to reach the stomach by applying it from this point along the back, passing along side of the ear to the front incisor teeth. At this point, which reaches the incisors, it is of assistance to make a mark on the rubber with a nitrate of silver point, this will avoid your pushing the tube out or in to discover whether the tube has reached the stomach after it is introduced.

In dilations and falling of the organ, the length of tube required can only be learned after a previous lavage. When the sound is used to draw out a test meal direct the patient to press in his abdominal muscles as if the act of having a stool. Frequently the accompanying nausea will bring this about involuntarily. If no contents arise, push the tube gently further in or pull it slowly out and try both ways. If the abdominal walls are flabby, external manual compression will sometimes produce the desired result. In all these manipulations are of no avail, the stomach is either empty or the tube is plugged up with food particles too large to pass.

To find out which is the case, allow 300 c.c. of pure water to run in. \times and then lower the funnel and siphon out, if nothing but comparatively clear water returns the test meal has passed into the duodenum. One should be very cautious in moving the tube out and in when no stomach contents appear in the funnel, as it is possible that the eyes of the tube may have sucked in the gastric mucosa itself and by moving too suddenly a piece may be torn away, if there is the least resistance avoid moving, rather pour in a small known amount

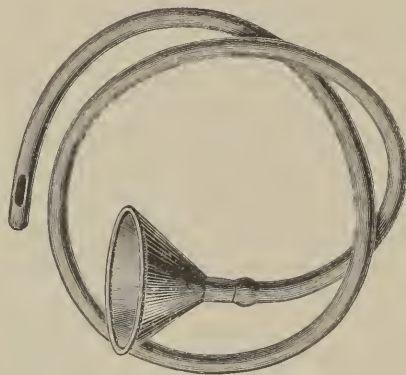


Fig. 8. EWALD STOMACH TUBE.

of water which will push away the adherent mucosa or the food particle and the next attempt will bring up the test meal.

If the stomach is already empty, the test meal must be given again at another time. I never recommend any apparatus for aspiration, not even the rubber bulb with patience the simple expression method will suffice. For small samples of test meals the Einhorn stomach bucket (Einhorn Diseases of the Stomach page 63) is an available instrument. I do not recommend the recurrent stomach tube, except as a time saver and am aware that its usefulness is very limited. Before using the tube, all artificial teeth should be removed.

In very rare cases of intense food and mucus putrefaction and in extensive gastrectasias it may be used with success. To give an idea of the time it takes to cleanse some stomachs, I quote Dr. Herman Strauss, assistant to Prof. Riegel who claims to have washed out rice particles after 40 litres of water had been allowed to flow in and out. After washing a dilated stomach for 1 hour personally, I found bread and stringy mucus in the last washing. Before introducing a stomach tube, assure yourself that its lumen is not obliterated and warm it in a pitcher of warm water. Dr. F. B. Turck, of Chicago, has devised a rubber pocket that is tied underneath the patient's chin to catch the saliva and mucus, etc., dropping from the mouth.

TEST MEALS.

The test meal most frequently employed is that of Ewald and Boas consisting of a roll or piece of wheat bread and 500 c.c. of water or tea, without milk or sugar. The time for examination is one hour after the meal.

Leube and Riegel advocate a test dinner of 400 c.c. soup, a portion of beef steak or roast beef, potatoes and a roll. The time for



Fig. 9. THE CÆSOPHAGEAL TUBAL PROBE.

examination is three to four hours after this meal.

Jaworski and Gluczinski employ hard boiled egg white and 100 c.c. water.

Klemperer recommended $\frac{1}{2}$ liter of milk and 70 grs. of wheat bread, and examined two hours later.

Germain Sée used 60-80 grs. scraped meat and 150 grs. white bread.—Examination two hours later.

The Ewald and Boas test breakfast seems the most convenient, and in cases of enfeebled digestion where much food is retained of previous meals, the least confusing.

At the Maryland General Hospital, we frequently use a double test meal consisting of—

At 8 A.M. 1 small piece of beef scraped and broiled = 80 grams.

1 of a soft boiled egg.

30 grs. boiled rice.

1 glass of milk and a piece of bread.

Four hours later an Ewald test meal is given, and, one hour after this the stomach contents are drawn. In giving a test meal, always insist on good chewing and urging all food substances to be very finely cut up so that they can not plug up the tube, even if not digested.

The double test meal, about which the late Dr. Henry Salzer was quite enthusiastic, really offers some advantages over others. In the first place it permits of as easy a study of the various stages of the digestion and of the motility and degree of retention as Riegel's test dinner; but the main advantage of the double test meal—a full meal at 8 or 9 A.M. and an Ewald test meal at 12 M. or 1 P.M., examination at 1 or 2 P.M.—is, that after drawing it, we may in a large number of instances recognize conditions of gastric motility and secretions before we analyze the contents. For instance; disappearance of the entire breakfast meal points to a normal digestion.

Absence of all proteids—beef and egg—and presence of considerable carbohydrates — rice and bread points to Hyperchlorhydria, and again;—Absence of all carbohydrates and presence of some of the beef and egg points to Hypochlorhydria, subacidity or anacidity. Presence of the entire meal with perhaps milk uncurdled—impaired motility with atrophy of gastric mucosa—absence of acid, enzymes and proenzymes. If the entire breakfast has disappeared, the status of the gastric secretions may be ascertained from the Ewald test meal which is still present.

The objection, which has been urged that the double meal is uncleanly to handle during analysis, is the same which was urged against Riegel's. Whether the morsels of an Ewald test meal are more appetizing and æsthetic to handle than remnants of our double test meal, is a matter concerning which it does not pay to quarrel.

It is a very important matter to state what test meal is used in giving out the various acidities obtained, because some test meals are greater stimulants to the gastric mucosa than others.

The Ewald test breakfast really makes very slight demands upon the working capacity of the stomach.

The total acidity one hour after an Ewald test breakfast is normally about 60;—the lowest total acidity observed by me one hour

after a test breakfast of this kind in a healthy individual was 22. Fleiner, who uses a test meal of soup, roast beef and potato puree, asserts that 3 to 3½ hours after this test meal, the total acidity is normally 70 – 100. (Prof. Wilhelm Fleiner, *Lehrbuch d. Krankheiten d. Verdauungsorgane*, p. 186). Dr. Julius Friedenwald has found that the gastric secretion of HCL appears sooner, and reaches a higher degree after our double test meal than after an Ewald meal.

An amount of HCL equal to 0.1 – 0.2% may be regarded as normal—everything below that means subacidity, above this, hyperacidity. The total acidity can not correctly be regarded as an unfailing indication of the amount of HCL present; the latter should always be determined separately in addition to the total acidity.

Apparently there are climatic, barometrical and geographical factors which influence the total acidity. In 170 cases at Riegel's clinic, Strauss found the total acidity after a test breakfast equal to 68; in 92 cases at Berlin after a test breakfast, the average total acidity was estimated at 47; the average amount of free hydrochloric acid at Riegel's clinic was found to be 37;—Normal values, one hour after a test breakfast of a roll and water are for average total acidity 40 – 60, for free HCL 20 – 30, for Baltimore.

After the complex meal of Salzer, *i.e.* 3 hours after, 50 – 60 grs. beef, 500 c.c. milk, 69 grs. rice and 1 egg, the total acidity on the average was found to be 95° and the free HCL 46°, for Baltimore. It should be emphasized that these figures represent only relative values. One often finds every symptom of hyperacidity with relief following the use of alkalies, when the total acidity was

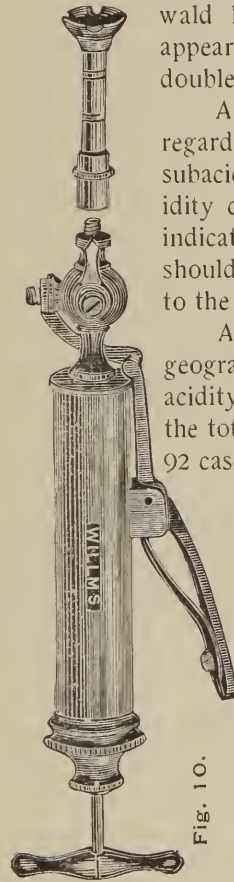


Fig. 10.

The Stomach Pump.

found to be only 56°, (1 hour after an Ewald breakfast) the free HCL only 24°; On the other hand, cases have presented themselves showing under the same conditions, a total acidity of 80° and free

HCL. = 40°—still no symptoms of hyperacidity. All this goes to show that some stomachs may do their work normally very well on relatively low amounts of free HCL, and, of course suffer from hyperacidity from comparatively slight increase of free HCL which would not affect a stomach used to higher amounts of acid.

Most modern observers that can speak with authority on the subject, agree that the total acidity should not be employed to express hyperacidity, but only the amount of free HCL, as this is the only acid which, when increased, gives rise to the complex of symptoms technically recognized as hyperacidity.

Before closing this lecture it might be added that where it is impossible to use the tube on account of prejudice of the patient, to



Fig. 11. MODIFIED EWALD TUBE.

obtain a test meal, emesis might be resorted to. The stomach contents obtained after a test meal, as a rule filter slowly; if much mucus is present, not at all. The filtration can be accelerated by rubbing the material first through a small coarsely grained sieve, (strainer) then through a finely grained strainer and then filtered through filter paper.

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LECTURE XIII.

METHODS FOR QUALITATIVE AND QUANTITATIVE ANALYSIS OF STOMACH CONTENTS — PRESENCE OF BITS OF GASTRIC MUCOSA — EXAMINATION OF STOMACH CONTENTS FOR MUCUS, SALIVA, BILE, DUODENAL SECRETIONS, BLOOD AND PUS — TESTS FOR BLOOD IN STOMACH CONTENTS — DEMONSTRATION OF THE PRESENCE OF IRON IN STOMACH CONTENTS OR VOMITED MATTER — SPECTROSCOPIC EXAMINATION OF STOMACH CONTENTS FOR BLOOD — EXAMINATION OF PORTIONS OF MUCOSA OR TISSUE FOUND IN THE WASH WATER OR VOMITED MATTER — LITERATURE.

THE STOMACH contents should be examined for—

1. The character and amount of the undigested food.
2. The presence and kind of bacteria.
3. The bile, mucus, pus and blood.
4. The total acidity.
5. The amount of free hydrochloric acid.
6. The presence of inorganic, lactic, butyric or acetic acids.
7. The combined hydrochloric acid and acid salts.
8. The presence of products of digestion viz: syntonin, propeptone albumoses, peptone.
9. The presence of pepsin and rennin.
10. The products of starch digestion, dextrin, erythrodextrin, achrodextrin and maltose,

PRESENCE OF BITS OF GASTRIC MUCOSA.

The examination of undigested food particles may demonstrate the presence of substances eaten 24 hours before the expression of contents and thus at once establish a dilatation or stenosis. As already pointed out, excess of rice and bread, and absence of beef and egg indicates a higher acidity, whilst absence of bread and rice, and presence of egg and beef indicates sub or anacidity; this of course cannot be seen unless the contents are drawn out about 3 hours after the complex meal, as employed at the Maryland General Hospital.

For bacteriological examination, a few slides are stained with methylene blue, and, also cultures made, the latter especially when there is any disease of the air passages, the microbes of which may get into the stomach with swallowed mucus or run down unconsciously during sleep. This is particularly important in pulmonary or laryngeal tuberculosis. Instead of methylene blue, Lugol's solution of iodine should be used on other slides, for examining bits of tissue, mucosa, cellular detritus.

The normal stomach contains many micro organisms, only very large numbers of bacteria have a pathological significance if by culture experiments they can be shown to be still capable of living. The presence of sarcinæ, biscuit shaped bacterial cells, generally occurring in groups of four, is generally looked upon as pathological.

Microbes only propagate luxuriantly when stagnation of gastric contents occurs, the secretory disturbances are then a secondary effect, a consequence of the stagnation. But primary reduction of HCL secretion has been known to cause a luxuriant gastric flora, since it is the HCL, which, to a great extent, inhibits their development and also destroys a large number of them. If there be no good peristaltic power, the diminution of HCL causes further disturbances in the stomach and intestines by accumulation of bacteria. But no degree of gastric acidity, no matter how great, can destroy all bacteria introduced.

Hyperacidity is as detrimental in its consequences as anacidity, because it inhibits normal intestinal digestion which is the best means of combatting fermentation and putrefaction. Hydrochloric acid undoubtedly inhibits or checks gastric fermentation to a certain ex-

tent, but all ferment producing microbes are not destroyed by it in the stomach, therefore one frequently finds gastric fermentation with hyperacidity of HCL and reversely fermentation may be absent where hydrochloric acid is entirely absent, when the motility is good.

This will again impress upon you the importance of an intact gastric peristalsis, for a certain time of action is indispensable for organized ferments to set up their characteristic decomposition even at the body temperature, with a good motility however the gastric chyme may reach the intestine, meeting a vigorous digestion before the bacteria get a chance to forge ahead of the normal unorganized ferments.

The most frequent of fungi in gastric contents is ordinary yeast and there should be no difficulty in recognizing it, unless occurring in very large numbers it has no pathological significance. Two more germs found in the contents are of interest—the sarcinæ and the Oppler-Boas bacillus which occurs in the gastric juice of carcinoma. Sarcinæ may be seen under the microscope without staining, they are indeed preferably to be examined that way as they stain so deeply with aniline dyes as to look like black patches.

Sidney Martin recommends dyeing a drop of stomach contents on the slide or cover glass and placing in a very dilute solution of gentian violet for 3 minutes, washing out in water and mounting in Canada Balsam. The gentian violet must be so diluted as to be nearly transparent. Yeast can similarly be stained by magenta or methylene blue solution (2%); If the latter is used, the preparation requires washing out in water.

Sarcinæ can hardly be said to have any pathological significance, according to Oppler, (München. Med. Wochenschrift, 1894, No. 29). They occur in ectasias occurring on a nonmalignant basis and in very atonic conditions, also in acute and chronic gastritis, in ulcer, in the gastric neuroses and the gastrophtoses.

Riegel agrees with Oppler in the assertion that sarcinæ are very rarely found in gastric carcinoma. They generally are observed in biscuit or bale-shaped groups of 4, 8 and 16, individual sarcinæ bunched together, their occurrence as single individuals is seen rarely.

The Oppler-Boas Bacillus (Oppler—zur Kenntniss d. Magen-inhalts bei Carcinoma Ventriculi, Deutsch. Med. Wochschr. 1895, Nr. 5.) is an unusually long and immovable bacterium, which was

observed in many cases of gastric carcinoma. In 20 cases of carcinoma, Kaufmann found these bacilli 19 times, and according to his investigations these bacilli have the power of abundantly forming lactic acid from various kinds of sugar. In the only case of the twenty just mentioned in which the Oppler—Boas bacilli were absent, the lactic acid was absent also.

According to Schlesinger and Kaufmann (Wiener. Klinische Rundschau 1895, Nr. 15.) the presence of a large number of these bacilli in the stomach contents is an indication of carcinoma, and their absence is of similar significance to absence of lactic acid. If a Stenosis of the pylorus is present then the absence of these bacilli is an argument against carcinoma. Riegel (l.c.) confirms the occurrence of these bacilli in enormous numbers in carcinoma, and adds that although there are numerous fungi that have the property of forming lactic acid in stomach contents, this can not alter the significance of the Kaufmann and Schlesinger observation. He does not consider these organisms as pathognomonic of gastric cancer, but as very important for the diagnosis.

Our knowledge concerning the bacteria occurring in normal and pathological stomach contents is very incomplete as yet. It appears however, that in all pathological processes we are not confronted with qualitatively new bacteria, but with excessive multiplication of those normally present. The disturbances produced by abnormal augmentation of bacteria in the stomach are explained by Minkowski (Minkowski über, d. Gährung im Magen.—Mittheilung. a. d. Medic. Klin. Königsberg, edited by B. Naunyn, Leipzig, 1888, S. 156.) in the following manner:—

1. Substances may be formed which irritate the mucosa, and provoke catarrhal inflammation.

- 2 Gas may be formed in considerable quantities causing distress by distention and increase the mechanical insufficiency already present.

3. The fermentation may give rise to toxins.

4. Putrefaction of albuminous bodies may produce alkaline bodies that will neutralize the hydrochloric acid or what little of it may yet be secreted.

5. Gastric fermentations may have a detrimental influence on the intestinal functions.

EXAMINATION OF STOMACH CONTENTS FOR MUCUS,
SALIVA, BILE, DUODENAL SECRETIONS,
BLOOD AND PUS.

The presence of mucus is evident to the naked eye by its stringy and tenacious character. Its chemical demonstration is carried out by dissolving the mucus in liquor potassa in which the mucin is soluble and from which it can be precipitated by acetic acid. When Pharyngitis, Laryngitis and Bronchitis can be excluded, then large quantities of mucus in stomach contents are indicative of gastric catarrh.

If the gastric contents consist largely of saliva, this can be demonstrated by the potassium sulpho cyanate, otherwise known as Rhodankalium, KCNS, which is a normal constituent of healthy saliva. Potassium sulpho cyanate gives a dark, purplish-red color upon the addition of a solution of chloride of iron.

Bile, if present to any considerable extent is noticeable at once to the naked eye by the yellow greenish tinge it imparts to stomach contents. Very slight amounts of bile and duodenal secretions are occasionally observed under normal conditions, particularly if the stomach be washed out early in the morning before breakfast, for there is no absolute closure of the pylorus when the stomach is empty.

Boas has however pointed out that constant presence of very evident admixture of bile and duodenal secretions points to stenosis of the descending portion of the duodenum. (Boas. *Deutsch. Med. Wochschr.*, 1891, No. 28., über die Stenose des Duodenum). As a rule, it will be necessary to assure oneself of presence of bile by the Gmelin test or the demonstration of bile acids or cholesterin.

Gmelin's test is carried out by adding 20 drops of fuming nitric acid to 10 c.c. of officinal nitric acid in a test tube. Ten c.c. of stomach filtrate are drawn into a pipette, and, holding the test tube with the HNO_3 in the left hand in a slanting, horizontal position, the urine is allowed to flow slowly from the pipette held in the right hand over the nitric acid. If the urine contains bile, there will be formed several characteristic rings of color, which, going from above downward, are (1) green, (2) blue, (3) violet and (4) red, but only the green color is an evidence of the presence of bile.

Better results are obtained by using a conical glass on a broad foot instead of a test tube. In the clinical laboratory they are of 60 – 79 c.c. or about 2 ounces in capacity. It is of small advantage to be able to place them alternately on and in front of a white and black background during the reaction. First, 20 c.c. of urine, if necessary previously filtered, are placed in the glass, then, 10 c.c. of nitric acid added by a pipette, which is carefully carried to the bottom of the vessel, here the nitric acid is very gradually permitted to escape by diminishing the pressure of the finger on the end of the pipette. In this manner it is easier to get the nitric acid under the urine. The display of the colors yellow, green, blue, violet and red occurs from above downward; the green color is the only one that is characteristic of bile elements.

The demonstration of the *bile acids* is done by first precipitating all albuminous bodies by boiling or by alcohol; a few drops of a solution of cane sugar are added and then drop by drop pure concentrated sulphuric acid; if the solution is now heated a beautiful purple-red color is obtained between 60° and 70°c (Pettenkoffer).

The presence of duodenal secretions is arrived at by testing the stomach contents for the specific ferment activity of trypsin, amylpsin and steapsin. (see pages 35, 36).

TESTS FOR BLOOD IN STOMACH CONTENTS.

Although blood may be present in the material drawn by a stomach tube, or vomit, it is not always easy to decide whether it was derived from the lungs or the stomach. Vomiting may produce a cough, and vice versa coughing may lead to an attack of vomiting, and in cases where either organ is liable to hemorrhage such as in tuberculous patients with a congestive state of the mucosa, it is in rare instances impossible to decide the origin of the blood.

In cases with copious and arterial gastric hemorrhage, the blood is bright red and clotted. A slower, but still quite profuse hemorrhage generally shows as a black clot, or mass of black clots. In very slow but continuous hemorrhage, the blood collects and may be partially digested or decomposed in the stomach before it is vomited as a black, coffee ground, material. The diagnosis of blood in the vomit is not always easily made.—There are four methods of

determining the presence of blood, and by one or more of them it will generally be accomplished.

The first is by the microscopical demonstration of the red blood corpuscles. In suspected cases of ulcer all vomited matter should be microscopically examined even when the blood is not evident to the naked eye.

The second is known as the Guaiacum test. Two or three drops of tincture of guaiacum are added to five c.c. of stomach contents in a test tube, and ether poured on the surface—if blood is present, a blue color develops where the two liquids meet. Equal parts of tincture of guaiacum and turpentine that have been exposed to the air may be used instead of ether. This test for blood is fallacious, as almost any carbohydrate—bile and saliva will produce the same color in the total absence of blood.

The Guaiac test, which was originally proposed by Almén and van Deen, becomes more reliable when executed by an improved method suggested by H. Weber. A considerable quantity of the filtrate is extracted or mixed with water; Glacial acetic acid, to the amount of $\frac{1}{3}$ of the entire quantity of water and filtrate mixture, must be added.

Of this acid extract, about 10 c.c. are poured off after settling, then 10 drops of tincture of Guaiacum and 20–30 drops of turpentine are added. If blood is present, the mixture becomes violet-blue, in case blood is absent, the color will be red-brown. The blue coloring matter that indicates blood, can be extracted by shaking the mixture with chloroform. Coffee ground vomit will not permit of the correct finding of blood with either the two preceding tests.

It may have to be differentiated from genuine tea or coffee vomit, or from bile by Gmelin's test. In this form of vomit, the corpuscles are disintegrated and the hæmoglobin transformed into insoluble hæmatin. Still there are two ways left to diagnose, the blood present, if any;—first the formation of crystals of hæmin and secondly, the demonstration of the presence of iron.

1. Preparation of hæmin crystals, 3–4 drops of the thick sediment is mixed on a glass slide with a little common salt, then 1–2 drops of glacial acetic are added and carefully heated over a small flame of a spirit lamp or Bunsen burner until bubbles begin to form. If blood is present on examining the preparation with the

microscope, reddish brown, oblong crystals of hæmin hydrochlorate will be recognized; their color, form and occurrence is characteristic. This test may fail in cases where blood is present.

2. Demonstration of the presence of iron; Naturally the patient whose stomach contents are to be examined must not have been taking iron in any form, nor any raw meats.

DEMONSTRATION OF THE PRESENCE OF IRON IN THE STOMACH CONTENTS OR VOMITED MATTER.

In case one is dealing with coffee ground material this test may become necessary. Some of the black sediment is placed in a porcelain dish, a few crystals potassium chlorate added and 2–3 drops of strong hydrochloric acid. On heating over a flame and addition of a few drops of a 5% solution of potassium ferro cyanide 4KCN , $\text{Fe}(\text{CN})_2 + \text{H}_2\text{O}$ prussian blue will be formed—Boas and Sidney Martin consider this a very delicate test. The prussian blue upon the occurrence of which this test depends is a complex cyanide of iron $4\text{Fe}(\text{CN})_3 \cdot 3\text{Fe}(\text{CN})_2$.

SPECTROSCOPIC EXAMINATION OF STOMACH CONTENTS FOR BLOOD.

A spectroscopic examination is possible when the red blood corpuscles have become dissolved, and the filtrate of the gastric contents contain oxyhæmoglobin. This compound of oxygen with hæmoglobin, is distinguished by two absorption bands in the spectrum which occur between the Fraunhofer lines *D* and *E* in the yellow and green. If after the recognition of these lines a reducing agent is added to the solution of oxyhæmoglobin, for instance, if it is shaken with ammonium sulphide, the two bands observed before, fuse into a single broad band, occupying the place of the two distinct and separate bands, or move beyond *D* toward the red of the spectrum. (Compare Eichhorst, *l.c.* page 523, also, Richard C. Cabot, Clinical Examination of the blood, Wm. Wood and Co., Publishers, New York, 1897, and, v. Yaksch, *l.c.*).

EXAMINATION OF PORTIONS OF MUCOSA OR TISSUE FOUND IN THE WASH WATER AND VOMITED MATTER.

In the wash water from almost every stomach also in the samples of test meals, gained by the Ewald expression method, and in vomited mater, small portion of the superficial mucosa of the stom-

ach, can on careful searching be found. Stimulated by reading the accompanying literature, particularly the work of Hayem, Boas, Einhorn and Cohnheim, I have during the last five years made a study of the tiny bits of mucosa.

To detect them more easily, the stomach is best washed in the morning before breakfast with 500 c.c. of warm water, which is poured into a shallow papier maché or hard rubber dish, the bottom of which is colored white and black; on this background the tiny bits of tissue from the mucosa, or from any neoplasm that may be in the stomach can be more easily recognized. These bits are of a reddish color usually, they may seem at times colorless, so that in a glass or pitcher they will be overlooked, on the dark flat dish they are quite apparent. These bits come from very superficial erosions, which are possibly caused by very slight local congestions or traumatism (Ewald *l.c.*).

It is conceivable that the contraction of the muscularis of the stomach may, if sufficiently powerful, effect an arrest of the flow of circulation in the folds and intense congestion of the veins and capillaries, which may give rise to small hemorrhages into the mucosa. These hemorrhagic areas are very poorly nourished by the blood current, and may eventually succumb to the autodigestive effect of the gastric juice, other gastric contractions then loosen, and cast off these tiny spots of necrosis (Hartung *l.c.*).

According to Virchow, (*l.c.*) circulatory derangements of the larger vessels of the stomach—the acute chronic gastritis especially, if accompanied with vomiting and colicky contractions are the cause of ulcers and erosions. Such erosions represent only the superficial stratum of the mucosa, generally only the vestibule or alveolus and the first third of the gland ducts, the entire lower half of the mucous membrane is not cast off. (Gerhardt, *l.c.*) The gland duct remaining shows nothing pathological. At the sides and edges, the glands become longer, and, the first ones that are intact usually curve themselves over the defect, partly covering it. Recovery takes place by the simple after-growth of the remaining portions of the glands.

In three stomachs, which were taken immediately after death, (not later than two hours after) I observed what was undoubtedly a superficial epithelial sequestrum resting loosely upon the mucous

membrane in many places of what I had every reason to believe was a perfectly normal stomach. The auto-digestion in this case, had been prevented by pouring 80% alcohol into the organ about 15 min. after death. In places, portions of mucosa half as large as a lentil seed, could be dislodged by a gentle stream of water from a wash bottle. The erosions include the inner third of the gland duct proper [Inneres Schaltstück of Stöhr] and it seems that even before they were dislodged, the process of repair had already begun. For underneath of small areas of necrosed superficial epithelium that was lifted from the true glandular stratum by a thin layer of lymph containing few red blood corpuscles, cell politeration was going on in the parietal or oxyntic cells, and in the cylindrical cells of the adjoining intact epithelium; formation of mitoses and karyokinetic figures are evident in picrocarmine and eosin stains of these sequestrations of mucosa.

It seems possible that a process of exfoliation is constantly going on in the lining membrane of the gastro-intestinal tract just as in the epidermis. It is not conceivable that the constant and continuous impact and friction with the ingesta should go on daily without causing necrosis of epithelium in places. If we should hold the normal acid chyme in the palm of our hands for 3-4 hours, three times or more every day, we would very soon notice exfoliations of the epidermis.

In the digestive tract—for it occurs all along the small intestine, this exfoliation goes deeper than in our hands because of immediate auto-digestion of the exfoliated spot. Although I have examined nearly fifty human stomachs with especial observation for this phenomenon I have failed to detect it in but four cases, and in these the examination was limited to a very small piece of stomach.

Even in stomachs obtained within one hour after death, and preserved by pouring alcohol or solutions of formaline into the organ, these erosions can be seen in places. I generally request a strip which begins in the œsophagus, runs through the cardia saccus coecus, entire greater curvature, pylorus and has a piece of duodenum attached to it. This is hardened and in many places pieces are excised half an inch apart and imbedded in celloidin, and, cut into serial sections with the revolving microtom, stained in eosin-hæmatoxylin and mounted in balsam. In some cases I sectioned strips

running along the lesser curvature.

In this way it was found that most of these erosions and exfoliations occur in the vicinity of the sphincter antri pylorici, about 7–10 cm. from the pylorus. At this point [see page 62] the muscularis has its most powerful development and the peristalsis and consequently the impact of the food with the mucosa is most vigorous, hence the epithelium here has most to suffer from friction. Slight erosions can be detected in the lower part of the oesophagus where no peristalsis occurs normally, but that accompanying deglutition. So the conclusion seems justifiable that very tiny exfoliations and erosions occur in all stomachs, and, in adult life perhaps at all times. This precludes the presumption that the pieces of mucosa are lesions produced by the stomach tube.

Boas [*l.c.*] thinks that coughing or defecation may cause the dislodgement of such loosened epithelium. When this process reaches such an exaggerated type as described by Einhorn, [*l.c. Erosions of the Stomach*] it is very probable that the mucosa is made less resistant by some well developed gastric disease, [one of the forms of gastritis, carcinoma, etc.,] for his patients suffered from pains, emaciation and weakness.

Among the forty-six stomachs examined by myself were nineteen in which no symptoms referable to the stomach were given during life. The pieces vary from to 5 mm. in length and nearly as wide. Einhorn recommends intragastric spraying of a solution of 1 per mille of argentic nitrate for the excessive exfoliation, combined with intragastric galvanization, diet and tonics, with a hygienic out door life.

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LECTURE XIV.

THE DIAGNOSTIC SIGNIFICANCE OF FRAGMENTS OF MUCOSA AND OF GASTRIC EXFOLIATIONS AND NEOPLASTIC TISSUE OCCURRING IN THE WASH WATER AND VOMITED MATTER.

BOAS, WHO first used these findings for diagnostic purposes attributed great importance to this way of finding out the real state of the mucosa. He held that in certain conditions of suppressed secretion, the differential diagnosis between a possible neurosis and a genuine gastritis with glandular atrophy was only possible by examination of such pieces of mucosa. Rosenheim *l.c.*, Boas *l.c.* and Julius Friedenwald, *Med. News*, June 22, 1895, emphasize the value of qualitative and quantitative testing of rennet zymogen to differentiate between chronic gastritis with glandular atrophy and carcinoma on the one hand, and nervous dyspepsia and secondary gastritis on the other. However, Ewald *l.c.* and also Einhorn *l.c.* have asserted that absolute deficiency of rennet zymogen is not pathognostic for atrophy, therefore it would indeed seem as if a certain diagnosis could only be made by a small piece of mucosa.

Is there any clue which can be derived from these pieces regarding the state of the mucosa in the secretory disorders? This I will try to answer in the following. Hayem to whom we are indebted for

the best histological investigations of the gastric mucosa, emphasizes that the individual elements of the mucosa, gland ducts, superficial epithelium and interstitial tissue can become diseased in a variety of ways, the various portions of the stomach fundus, pylorus and cardia may exhibit different affections; and finally the mucosa may at different parts show different phases of disease. He distinguishes a parenchymatous and an interstitial gastritis.—First the parenchymatous.

1. Gastrite parenchymateuse hyperpeptique chloro-organique.

Under this he has two subclasses:—(a) D'emblée—coming on at once—in the first stage of digestion. (b) Tardive—coming on in later stages—in 1½ to 2 hours. Under this hyperpeptic parenchymatous gastritis, Hayem means clinically, a hyperpepsia with hyperacidity and anatomically, degeneration of the principal central or chief cells with proliferation of the parietal border or oxyntic cells.

2. Gastrite parenchymateuse muqueuse=gastritis mucipara, by which he means a mucous degeneration, a process taking place principally in the vestibules to the gland ducts (which are lined with columnar epithelium) and corresponds to the *Schleim Katarrh* of most German writers. This is associated with hypopepsia and subacidity.

3. Gastrite parenchymateuse atrophique, which signifies anatomically the total atrophy of the glands—without interstitial processes—and clinically—Anacidity or Achylia. The interstitial forms he separates into two classes.

a. Those in which the round cell in filtration.

b. Those in which the Sclerosis *i. e.* connective tissue proliferation predominate. These processes are described as occurring purely as such or mixed with forms of parenchymatous gastritis and as leading to sub or anacidity. In order to bring my results in critical consideration with those of Einhorn *l.c.*, I have adopted his classification of the anatomical conditions found in these fragments. There is however one objection that can be urged against it and that is the apparent fact that he has based his system upon conditions of the gland tubes and interglandular tissue exclusively and mentions the state of the cells only once in six types described. I will therefore supplement his categories by adding the state and condition of the

vestibular or alveolar columnar cells (Vorraum Zellen) and the condition and numerical relations of the chief, central or ferment cells (Hauptzellen) and the parietal, border or oxyntic cells (Belegzellen).

1. Normal—Gland ducts and interglandular tissue exist in normal proportions. Columnar epithelium of the surface and that of vestibule normal with scarce cells showing at their free ends slight mucoid metamorphosis. Average number of parietal or oxyntic cells in 6 ducts which were sectioned very nearly down the centre = 22—40.

2. Connective tissue excess—Proliferation of connective tissue around the glands—glands and epithelial cells as in normal condition.

3. Proliferation of Glands. Under this class I have in nineteen cases been impressed with the probability that there must be three types of this condition.

Type *a* In this subtype there is a proliferation of gland tubules. Under the same field of microscope there will be more than under normal conditions, since they are much closer to each other, but the number of central and oxyntic cells are from 18—42 or the same as under the normal condition.

Type *b* Increase of oxyntic or parietal cells with normal number of gland ducts. Here there seems to be no proliferation of the gland ducts—the connective tissue and the ducts bear the same relation as in class 1, but the aniline staining, oxyntic cells may be so increased that they lie in juxt apposition giving the whole duct the appearance of a peptic duct of the dog; the number may reach 70 in one duct. The oxyntic cells are increased in size.

Type *c* Increase of the number of ducts in which the number of oxyntic cells appear normal in size and number, and, in the same fragment or section portions of mucosa in which the ducts are not augmented but the oxyntic cells are increased in number and size; this third is then it would seem a combination of types *a* and *b*. When there are many oxyntic cells above the normal the entire gland duct assumes a tortuous or elongated shape. It seldom extends down into the mucosa in the same plane, therefore it is very rare that a section will strike down the middle of a duct. Generally the counts in six ducts struck fairly along the central canaliculus are taken

as an average.

4. Incipient Atrophy—To the same field under the micrometer there are fewer glands than normally present, they appear shrunken and smaller, at the same time the spaces between the glands are larger than normal owing to an increased connective tissue formation, the latter is thickly invaded as a rule, with small round cell infiltration. The next type is:—

5. Atrophy—In complete atrophy there are only remnants of glands left, a few degenerated cells lying in empty circular spaces where glands had previously existed, there is also a diffuse round celled infiltration.

6. Vacuolization.—Round or ovoid vacuoles exist within the glands in large numbers, being the result of mucoid degeneration of some of the glandular cells, this is generally associated with connective tissue proliferation. Vacuoles are present in the gland cells normally and can be seen in the drawings of Kupfer and Stöhr. I have also seen them in both longitudinal and cross-section of the gland tubules but rarely more than 2 to 3 to the entire duct. It is conceivable that they may be produced by the process of hardening and imbedding. Some of the fragments obtained from stomachs may show characteristics of two types.

DEDUCTIONS FROM 36 CASES.

In eight healthy persons the mucosa fragments were normal in six. Proliferation and autodigestion marked in one, which showed also beginning small round cell infiltration between the ducts—connective tissue increase in one. In the first of these cases the examination showed proliferation in one fragment and a normal condition in a second one found in the same wash water.

In eighteen cases of hyperacidity, the fragments of gastric mucosa found were apparently normal in four.

Atrophy of gland tubules and connective tissue increase, so that there were fewer glands, but in these few there were contained a larger number of oxyntic cells than normal in two cases.

Proliferation of gland ducts with apparently normal oxyntic cells in six cases.

Proliferation of oxyntic cells, generally without marked increase in the gland tubules in six cases.

In twelve cases of anacidity or subacidity the fragment was apparently normal in two cases.

Proliferation of glands with marked small round cell infiltration was found once.

Atrophy in some form was found in the fragments from the nine remaining cases.

In establishing the classification of euclorhydria and hyperclorhydria we could not be guided exclusively by the amount of free HCL found after the double test meal.

Thus, a young vigorous farmer, aged 25, who never had any disease showed on repeated examination an amount of free HCL equal to 60° with a total acidity of 80°. Ordinarily judging simply from the analysis, such a case would be diagnosed as hyperacidity, according to the principles defined on pages 89 and 90; however, these cases can be diagnosed justly and accurately, when considered together with concomitant signs and symptoms only. Although this case had the large amount of free HCL, there was no starch indigestion, no erythrodextrin, no pyrosis; there were no symptoms referable to the stomach at all, the man was in perfect health.

Another case, a neurasthenic female had intense suffering from hyperacidity and occasional gastroxynsis and the amount of free HCL was never over 40°. This case showed hypermotility, the stomach as a rule, was empty 25 minutes after an Ewald test meal; with my intragastric rubber bag in connection with the kymograph. she showed very frequent and sudden gastric peristalsis of unusual tonicity.

Summary.

8 healthy persons:	{	Perfectly normal in 6. (a) Glandular proliferations, (b) Normal in 1. Connective tissue increase in 1.
Hyperacidity in 18 cases:	{	Normal in 4. Atrophy in 2. Proliferation of glands in 6. Proliferation or hypertrophy of oxyntic cells in 6.
Anacidity or Subacidity:	{	Normal in 2. Proliferation of glands in 1. Atrophy in 9.

Proliferation therefore, according to this table is present in $\frac{2}{3}$ of these cases of hyperacidity and atrophy in $\frac{3}{4}$ of these cases of

anacidity or subacidity. Einhorn *l.c.* does not give any results from examination of perfectly healthy individuals as his cases of Euchlorhydria seem to be in patients.

Of his 12 hyperacid cases three were normal or very nearly so, six showed proliferation and three showed connective tissue proliferation. In his cases of anacidity or rather what he calls achylia gastrica of which there were seven cases, there was atrophy 3 times; marked vacuolization once, proliferation once, and normal condition twice.

On the whole, judging from Einhorn's results, Cohnheim's, Hayem's and my own, the conclusions seem justifiable that proliferation of glandular elements is present in from $\frac{1}{2}$ to $\frac{2}{3}$, the cases of hyperacidity and atrophy is present in from $\frac{1}{2}$ to $\frac{2}{3}$ of the cases of anacidity.

Adolf Schmidt, (*Virchow's Archiv*, Bd. cxliii, S. 478.) asserts that the epithelium of the surface of the stomach is preserved better than the gland cells in inflammatory conditions of the mucosa, this he says is particularly so in chronic gastritis which forms island like foci in stomachs otherwise not much changed. My experience and that of W. D. Booker, is not in accordance with this observation (see pathology of simple acute and chronic gastritis in the clinical portion of this work). Although I preserved the stomachs by injecting them immediately after death (within 20 min.) with alcohol, also with formaline and sublimate so that autodigestion was at once checked, my sections showed generally a more serious destruction of the surface epithelium than of the gland cells. At times both are so much altered that it is impossible to say which is most or least affected. It seems in chronic gastritis that new epithelium will be reformed quite rapidly where the old has been lost or destroyed.

In suspected cases of malignant neoplasms fragments of the growth are occasionally found and are of importance in the diagnosis. In carcinoma of the cardia or the oesophagus they are most frequently found in the lower or side opening of the tube as it must pass through or over the growth on its way into the stomach. But even in malignant growths of other parts of the stomach, patient searching in the sediment of the wash water will sometimes reward the clinician by the discovery of tumor fragments. The first wash water in the morning about 500 c.c. should be permitted

to settle 12 hours in a conical glass such as is used for the settling of urinary solid constituents, the sediment should be examined under a low power (about 50 diam.)

Once I made the diagnosis of carcinoma when no tumor was evident from repeatedly finding involuntary muscle fibres when no meat had been eaten for 3 days after preceeding lavage. It proved to be a broad flat carcinoma of the posterior wall.

The accompanying plates and drawings of normal and pathological gastric mucosa were executed by myself. The drawing showing a longitudinal section of the secreting gland tubules showing beautifully the well preserved cylindrical epithelium of the gastric surface and well differentiated oxyntic and chief cells was made from several sections of a piece of mucosa that was torn loose by the stomach tube, by a medical student who tried to aspirate a meal that had disagreed with him by means of the pump. The tearing off must have occurred in an instant as there are no signs of inflammation in the sections. The sections were stained in a variety of ways principally in the eosin-haemotoxylin, Golgi and Bismark brown stains. The minute communications of the oxyntic or parietal cells with the central duct are best brought out by the Golgi method.

The drawings of fragments found in the wash water of glandular proliferation with glands closely packed and connective tissue diminished and of glandular atrophy, mucoid degeneration, vacuolization and small cell infiltration are all explained by the text accompanying the illustrations. We have seen that histological changes approaching or actually representing pathological states may be going on in perfectly healthy stomachs. Furthermore, the stomachs of diseased patients may on serial sections, show a different pathological state at different places of the mucosa. Therefore it must be borne in mind, that although the findings in hyperacidity and anacidity appear to be in some relation to the disease; this kind of investigation must not be relied upon as representing in a given fragment the condition of the entire mucosa. It represents the state of the location from whence it sequestered, that not being accurately known generalizations must be made with caution.

LECTURE XV.

OCCURENCE OF SECRETIONS IN THE EMPTY STOMACH
— STIMULATIONS TO SECRETIONS OF GASTRIC JUICE
— SIGNIFICANCE OF FOAM —*PREPARATION OF
GASTRIC CONTENTS — QUANTITATIVE ANALYSIS — METHODS — STANDARD OR
NORMAL SOLUTIONS — INDICATORS
— TITRATION — APPARATUS.

MOST AUTHORS are of the opinion that no secretion is contained in the empty stomach. Schreiber (*Arch. f. exper. Pathol. u. Phar.*, Bd. xxiv, S. 365, also *Deutsche med. Wochschr.*, 1894 No. 18-21.) however, concludes that a secretion is found also in the empty stomach, that is he denies a continuous secretion or gastro-succorrhoea as a disease *sui generis* and claims to be able to obtain 60 c.c. of a secretion possessing good digestive power, from a je-june, fasting stomach before any food has been taken.

Pick, (*Prager med. Wochschr.*, 89. N. 18) who obtained similar results believed that the secretion was set up by the stimulation of the sound used. Rosin, (*Deutsch., med. Wochschr.*, 1888, No. 47,) A. Hoffman, (*Berlin. klin. Wochschr.*, 1889, No. 12,) and Martius, (*Deutsch. med. Wochschr.*, 1894, p. 638.) have also obtained a digestive secretion from the fasting stomach.

*The section on Quantitative Chemical Analysis of Gastric Contents has been written by my associate, Dr. Edward L. Whitney, whose experience as demonstrator of Clinical Pathology has admirably fitted him for the concise and clear account of this department. It gives me pleasure to express my thanks to him for his assistance.

Although there may be found 50–60 c.c. of a secretion capable of digesting, in the empty normal stomachs of perfectly healthy individuals, this does not prove that a continued secretion exists normally. Riegel, (*Deutsch med. Wochschr.*, 1893, p 735.) Leo (*Krankheiten d. Bauchorgane* p. 54) considers this digestive secretion a residuum of the last, previous meal and seems to have shown conclusively, that such a residuum is constantly present in the stomachs of infants after a night's sleep (See Leo—*Berlin Klin. Wochenschr.*, 1888 No. 49.) For the practical objects of diagnosis he concludes that a secretion of 50–60 c.c. of digestive fluid found in a fasting organ must not be considered pathological. Only when the amount gained reaches 100–300 c.c. it indicates hypersecretion which is often associated with hyperacidity. Reichmann (*Berlin Klin. Wochschr.*, 1887 s. 12.) Bouveret *l.e.* Débove and Rémond (*Les Maladies de l. Estomac*)—Riegel and Reichmann do not distinguish sufficiently between so-called continuous secretion of gastric juice with a stomach of normal capacity and normal exit to the duodenum and continuous secretion which appears as a concomitant symptom of Gastrectasia with probable pyloric stenosis. Einhorn, with more accurate differentiation between these states, it will probably be found that the normal stomach in a fasting state, contains very little if any secretion. I have seen a number cases whose stomachs were of natural size and where there was no disturbance but which contained this secretion early in the morning before breakfast.

J. Schreiber (*Deutsch med. Wochschr.*, 1894, No. 53.) has experimented upon two healthy persons and found gastric juice with hydrochloric acid in both, before any food had been taken. The amount of secretion thus obtained varied from 10 to 22 c.c. Martius (*Deutsch med. Wochschr.*, 1894, 32.) and Huber (*Korrespondenzblatt f. Schweizer Aerzte* 1894, No. 19.) confirm Schreibers results. According to Ewald, who sums up the Literature, (In Lubarsch and Ostertags, *Ergebnisse d. Speciel. Pathologie*, Bd. 3, S. 27.) and gives his own observations in a large number of cases, this problem is represented in the following manner. In many individuals small quantities of a digestive secretion containing free hydrochloric acid can be obtained from the fasting, jejune stomach, Sometimes it is mixed with bile coloring matter and duodenal contents. But he claims that the stimulation to this secretion has been furnished by

swallowed saliva, (*Martius*) remnants of food, pharyngeal secretion, etc., and that the state of things lies between a normal and an abnormal one, and that there is no diseased condition of the gastric mucosa.

In case of typical Gastrosucorrhoea however, there is a much increased irritability of the mucosa giving rise eventually to a profuse secretion, which, when found in empty stomachs, is quantitatively more considerable than that found in normal jejune stomachs. Huber compares it to a slow, gradual, dying away of the tonus of secretory irritability (*Abklingen des Sekretions reiz*) that has been set up by the ingesta and seems to linger after they have passed into the duodenum.

In order to obtain gastric secretion a variety of methods have been suggested.

By chemical stimulation, according to Leube's method which consists in allowing 50 c.c. of a 3% solution of sodium bicarbonate flow into the stomach. After 12 minutes this is washed out again and should be found neutral. By thermic stimulation according to Jarowski's method, consisting of the introduction of 100 c.c. of ice water and washing it out again after ten minutes when it should contain acid and pepsin. These methods if successful at all, bring out the gastric juice in a most diluted state and therefore give no adequate means to determine the secretion by chemical analysis. It has been claimed by Einhorn (*New York med. Record*, 9 Nov. 1889) and Allen A. Jones (*Ibid* 1891) in this country, and, Hoffman (*Berlin klin. Wochschr.*, 1889, N. 13) Ewald *l.c.* and Ziemssen, in Germany that the gastric secretion, as evinced by the amount of hydrochloric acid could be increased by faradic or galvanic stimulation. Whilst I have my doubts about this matter I do not wish to imply that electricity is not a very valuable therapeutic agent in the treatment of digestive diseases; we could not in fact dispense with it as an auxilliary to treatment. In my opinion the influence of electricity on secretion is not yet satisfactorily proven.

As a means to obtain gastric secretion this method is certainly not available. The normal secretions are best obtained by the natural stimulation of one of the test meals given on page 87 and 88. Among the list there mentioned the test meal of Fleiner should have been stated which is similar to Riegel's test dinner and consists of a plate of soup a portion of tender beef steak or roast beef and po-

tatoe purée.

Mathieu and Rémond [*Société de Biologie* 1890] have published a method to determine the total quantity of stomach contents by finding out the acidity of the undiluted contents as much as can be drawn then that of the contents as much as can be gained by washing out the stomach with a known quantity of water and from this the acidity of the total amount of contents that were originally in the stomach are calculated. Strauss (*Therapeutische Monatshefte* März 1895), has simplified this procedure, but for the practitioner it is sufficient to know the amount gained by the simple methods of drawing the contents by expression or aspiration. Concerning the recognition of proteid and carbohydrate indigestion from the food remnants it should be added this is much facilitated by the double test meal used at our hospitals.

In Gastrectasias presence of foam indicates gas fermentation. Gas may be found even in presence of normal or supernormal amount of hydrochloric acid, since F. Kuhn (*Zeitschr., f. klin. Medizin* Bd. xxi and *Deutsch Med. Wochschr.*, 92, No. 49) has demonstrated that the hydrochloric acid of gastric juice has no detrimental effect on large amounts of yeast. Whenever there is stagnation of gastric contents this gas formation can occur.

After the contents of the stomach are withdrawn, they must be prepared for and submitted to chemical examination. The contents may be beaten up thoroughly to make a homogeneous mixture and the chemical examinations conducted on this mixture, or this mixture may be filtered and the clear filtrate subjected to analysis. The former method gives the more accurate results, with slightly higher acidity, than the latter method; which has the advantage however of allowing better observation of color changes in the solution during titration.

Before entering upon a discussion of the chemical methods as applied to the gastric juice, a short description of the methods, solutions and apparatus required in quantitative analysis will be given.

The solutions required can be made up, and if preserved from the influence of light and air, can be kept indefinitely.

The methods used in quantitative chemical analysis may be divided into two general classes; Gravimetric and Volumetric. The

Gravimetric methods consist of the isolation of the substance or one of its compounds which is weighed. The isolation of substances in a pure state often requires long training in chemical methods, and if a small amount of the substance in question is present it may be very difficult to separate a weighable amount unless large quantities of the mixture are available. Many substances can not be separated from mixtures without losing at the same time their relation to other substances in the same solution. The great objection to the Gravimetric system however is the large amount of costly apparatus necessary, and the length of time needed for the manipulations.

The Volumetric methods are more easily performed, in this, the quantity of the substance under examination is ascertained by a calculation based upon a measured quantity of a solution of a known strength required to perform a certain reaction with it. These solutions called standard solutions, are of two kinds, normal solutions and Empirical solutions.

A Normal Solution is one which contains in a litre, a quantity of the active reagent, expressed in grammes and chemically equivalent to one atom of hydrogen.

Decinormal Solutions, N_{10} , are one tenth the strength of normal solutions.

Centinormal Solutions, N_{100} , are one hundredth the strength of normal solutions.

Empirical Solutions are those which do not contain an exact atomic proportion of the reagent, but are made up of such strength that one c.c. is equivalent to some definite weight of the substance sought.

Residual titration or back titration consists in treating the substance under examination with standard solution in excess of that known to be required, the excess is then ascertained by residual titration with another standard solution.

In general, titration results in the formation of a compound that can be distinguished by its properties, from those substances present in either solution.

1. It may form a precipitate.
2. It may cause the complete solution of some precipitate.
3. A slight excess of either reagent, may produce some visible

change in some constituent of the solution, or a change in some substance added for the purpose. (Indicators.)

4. The indicator in some cases can not be added to the solution but from time to time a few drops of the solution are added to the indicator on the side.

Of the above solutions, the normal solution is the most used, the empirical solution being only of limited application.

It would seem a simple matter to make up a standard solution which would be perfectly accurate, but the problem is not so simple. Absolutely pure chemicals are not easily obtained, and such as are easily obtained, unmixed with other mineral substances, contain a variable amount of water, and are moreover exposed to more or less danger of contamination from the impurities of the air. The following methods of obtaining a tenth-normal solution are recommended as a basis for the preparation of other solutions.

1. Pure, dry, oxalic acid is obtained and the crystals that show no sign of efflorescence selected. From the formula, $C_2 H_2 O_4 + 2 H_2 O$, it is seen that the molecular weight is 126, and as it is a dibasic acid, the normal solution would contain one half of this, (63 grammes) dissolved in distilled water and made up to one Litre, at a temperature of $15^{\circ}C.$, As a tenth normal, N_{10} , solution is required; one tenth of this or 6.3 grammes are made up to a litre as before and used to correct the solutions used in analysis. It must be noticed that oxalic acid in dilute solution soon decomposes so that it must be freshly prepared as required.

To prepare an equivalent solution of caustic soda (decinormal NaOH) about five grammes of caustic soda are dissolved in about 800 c.c. of distilled water and well mixed. To this there is added lime water or baryta water $Ca (OH)_2$ or $Ba (OH)_2$ as long as a precipitate forms, to get rid of carbonates or sulphates. The solution is allowed to stand until the impurities have settled. Twenty-five c.c. of the solution are then measured with a pipette into a clean flask or beaker and titrated with the above solution of oxalic acid, using a few drops of phenol phthalein as an indicator, until the red color of the solution just disappears. The solution is then diluted to the strength of a decinormal solution.

As an illustration of the method of ascertaining the amount of dilution necessary to make the two solutions exactly equivalent, we

will suppose that the twentyfive c.c. of caustic soda solution required 28.3 c.c. of the oxalic acid solution to discharge the red color. If twentyfive c.c. of the caustic soda solution neutralize 28.3 c.c. of the acid solution, then the amount of caustic soda solution necessary to neutralize 1000 c.c. of the acid solution will be found by the following proportion.

$$28.3 : 25 :: 1000 : (x) \qquad x = 883.4$$

x = amount of of caustic soda solution necessary for 1000 N₁₀ NaOH. Dilute 883.4 c.c. of the caustic soda to 1000 c.c. with distilled water.

After diluting the solution it should be again titrated to ensure its accuracy, and if properly standardized it should be changed from red to colorless and *vice versa* by the addition of a drop or two of the acid or alkaline solutions respectively. The titration should be conducted as rapidly as possible to avoid the error produced by absorption of CO₂ from the air and all solutions kept in well stoppered bottles for the same reason.

2. About eight grammes of pure dry sodium carbonate are heated in a platinum crucible for ten minutes at a *dull red* heat, stirring occasionally with a platinum wire. After heating, it is powdered in a warm mortar and allowed to cool in a dessicator. When cool, 5.3 grammes of the powder are weighed rapidly, washed into a flask with hot distilled water and made up to a litre. This constitutes a decinormal solution of sodium carbonate.

A decinormal solution of sulphuric acid is prepared in the following manner. About three c.c. of the pure strong acid, of a specific gravity of 1.840, is made up to about 900 c.c.

This approximate solution is standardized against the sodium carbonate solution prepared as above, using a drop or two of a—0.1% solution of methyl orange as an indicator. Twenty-five c.c. of the acid solution is titrated with the decinormal sodium carbonate until the red color shown by this indicator in acid solution turns to a light yellow. The correction of the approximate solution is made from a proportion upon exactly the same principle as in the former case (No1.)

To correct this decinormal solution of sulphuric acid for very accurate work, the following method is recommended. One hundred c.c. of the decinormal solution of sulphuric acid is alkalinized

with a strong solution of ammonia— (ammonium hydrate.) The solution is evaporated to a constant weight on the water bath and the amount of sulphuric acid calculated from the amount of ammonium sulphate formed.

Indicators.— An indicator is a substance used in volumetric analysis which indicates by change of color or some other visible effect, the exact point at which a given reaction is complete.

Generally the indicator is added to the substance under examination, but in a few cases it is used, outside, a drop of the substance being brought in contact with a drop of the indicator.

The particular uses of the indicators will be more fully explained in their proper places, under the quantitative examination of the gastric juice, but the chief ones in use in such examinations may be briefly mentioned.

Tincture of litmus which turns red in acid solution, blue in an alkaline solution. It is used in solution, and also in the form of test-papers. (It is not used when carbonates are present.)

Phenolphthalein solution, a 1% solution of phenolphthalein in alcohol, colorless in acid solutions, red in alkaline solutions. Is not reliable for alkaline phosphates, bicarbonates, or ammonia.

Methyl orange solution, a 0.1% solution of methyl orange in water turns red with acids, yellow with alkalies. It is not affected by carbonic acid, and is valuable for titration of alkaline carbonates.

The other indicators and their uses in analysis of the gastric juice will be mentioned later.

Apparatus.— The apparatus needed for volumetric work is comparatively simple. Burettes, measuring flasks, measuring cylinders, and pipettes. An accurate balance is required in all chemical work, delicate to a milligramme and weighing up to say fifty grammes. Burettes, are glass tubes graduated to tenths of a c.c. and holding from 25 to 100 c.c. They are provided at the lower end with a rubber tube and pinchcock by means of which the amount of the solution can be accurately regulated. The tube is graduated upon its outer surface and the amount of the solution used, can be read off from this graduation. The simplest form of burette is the one already described, known as Mohr's, of which various modifications are in use.

Measuring flasks are vessels made of thin glass, having a narrow neck, and so constructed that a certain amount of fluid reaches a graduation placed about the middle of the neck. These flasks are of various sizes, 100, 200, 250, 500 and 1000 c.c.

Measuring cylinders, are of various sizes, from 25 to 1000 c.c. graduated from 0.5 to 5 c.c.

Pipettes are of two kinds, those graduated for one quantity only, and those graduated on the stem to deliver various quantities. A convenient set for stomach work is the following 2, 5, 10 and 25 c.c.

In addition to the above apparatus, one should be supplied with funnels, crucibles, beakers, flasks, test tubes, Bunsen burners, etc.

All apparatus should be kept scrupulously clean, rinsed before and after using with distilled water. It is well to wash the inside of any measuring apparatus two or three times with small portions of the solution for which it is to be used.

Greasiness interferes very much with accurate reading. It may be removed by dilute alkaline solution.

The burette should be placed perfectly perpendicular, and firmly fastened. Fill by a funnel, the stem resting against the inner surface of the burette, to avoid the formation of bubbles. Always fill above the zero-mark, gently tap the burette until the bubbles disappear should they be formed. Then run out a small portion (or down to the zero mark) remembering to run out enough to remove all air bubbles from the bottom of the burette.

In reading the results, always read from the bottom of the meniscus, formed by the rising of the outer borders of the liquid along the sides of the burette.

LECTURE XVI.

CHEMICAL EXAMINATION ON GASTRIC JUICE — TESTS FOR PRESENCE OF FREE ACIDS — TESTS FOR FREE HYDROCHLORIC ACID — THE DIM-ETHYL-AMIDO-AZO-BENZOL TEST — THE RESORCIN TEST — COMBINED HYDROCHLORIC ACID — LACTIC ACID: FORMATION, SIGNIFICANCE, DETECTION.

REACTION: The reaction of the gastric juice, obtained by means of the stomach tube or otherwise, after the administration of a test meal, is always acid in the normal individual. The reaction is best determined by dipping into the juice, a piece of very delicate blue litmus paper. In juice of acid reaction the paper immediately turns red. Very rarely is the reaction alkaline, this being found only in a few cases of atrophy of the gastric mucosa, occasionally in acute gastritis, and when for some reason, a portion of the intestinal contents and the alkaline bile has been forced back through the pylorus in sufficient quantity to neutralize the acid of the stomach.

In severe cases of gastric atrophy the reaction is usually acid, even in absence of fermentative changes. This is due to the presence of acid salts, such as acid sodium phosphate ($\text{Na H}_2\text{PO}_4$), and of traces of organic acids, which occur in nearly every test meal in quantities sufficient to produce an acidity of from 6 to 10 degrees.

TESTS FOR PRESENCE OF FREE ACIDS.

A delicate test for the presence of free acids is found in Congo red. Congo red occurs as a fine reddish brown powder, dissolving readily in water to form a clear deep red solution, which changes in the presence of free acids to a dark blue. This substance may be used in two ways as an indicator.

1. A solution is prepared by dissolving one grm. of the powder in 100 c.c. of water, and adding a small drop to a few c.c. of the gastric juice. If the juice contains even a slight trace of free hydrochloric acid, or the organic acids in slightly larger quantities the solution immediately turns a bright blue.

2. A test paper may be prepared by soaking bibulous paper in the above solution of the dye for several hours and then carefully drying. This paper is simply dipped into the filtrate or into the contents before filtration and exhibits the same color reaction as the solution mentioned above, and has the additional advantages, of being more convenient, and, exhibiting as readily slight changes in color. It has been found also, that when the acidity is due to organic acids and not to free hydrochloric acid, the color can be made to disappear by warming gently over the open flame. On the contrary if the acidity is due to hydrochloric acid, the dark blue stain on the paper changes to a lighter tint but does not disappear except when strongly heated.

It must be emphasized that this color change from red to blue does not occur in solutions of acid salts or in the presence of combined hydrochloric acid and therefore indicates the presence of some free acid, inorganic or organic. •

TESTS FOR FREE HYDROCHLORIC ACID.

Many tests have been proposed for free hydrochloric acid, the following given in the order of their accuracy and delicacy being probably the most reliable.

- | | |
|------------------------------|------------|
| 1. Dimethyl—amido—azo—benzol | 0.02 p. m. |
| 2. Phloroglucin—vanillin | 0.05 p. m. |
| 3. Resorcin | 0.05 p. m. |

THE DIMETHYL—AMIDO—AZO—BENZOL TEST.

This test, recently introduced by Töpfer, is probably destined to replace all others in the clinical laboratory, both on account of its simplicity and also on account of its ready application to the direct quantitative estimation of the amount of free hydrochloric acid in the gastric juice. This indicator occurs in the form of a brown powder, readily soluble in alcohol, only slightly soluble in water. A few drops of the alcoholic solution, added to a solution of hydrochloric acid turns a bright cherry red, increasing in intensity as the strength of the acid solution is increased. In the absence of free hydrochloric acid or other mineral acid the solution turns a bright lemon-yellow.

In actual practice a 0.5% solution of the substance in alcohol is employed. A few drops of this solution are added to the stomach contents which need not be filtered for this purpose, or to the residue left in the receptacle in which the stomach contents were received. If free hydrochloric acid is present the cherry red color develops and spreads in beautiful rings from each drop of the indicator, usually leaving in the center a clear yellow area. In case the indication is doubtful the following modification may be employed. A small porcelain evaporating dish (or white butter plate) is thoroughly rinsed with distilled water and dried. Upon one side of the dish a few drops of the filtrate are placed and upon the opposite side a single drop of the indicator. By inclining the dish gently the two solutions may be made to mix and, at the line of junction, the cherry red color may be seen, the white back ground rendering the detection of the tint less difficult.

It has been stated by Einhorn, and others that this test is liable to mislead in cases in which there is a large amount of organic acidity. It is true that in the presence of lactic acid amounting to 0.2% or more in gastric juice this test yields a red color resembling that due to inorganic acids but the objection is more theoretical than real, as the presence of such an amount of organic acids seldom occurs in the stomach and in the presence of proteids, peptones mucin etc, still stronger solutions of the organic acids are required to produce the characteristic reaction.

Futhermore the quantitative estimation of organic acidity to be described presently will show the necessity of employing further

tests for the presence of free hydrochloric acid, on account of a specially great acidity of organic acids, which does not occur in a stomach secreting a normal amount of hydrochloric acid.

THE PHLOROGLUCIN VANILLIN TEST.

The modification of this test proposed by Boas gives the most satisfactory results. Two grammes of phloroglucin and one gramme of vanillin are dissolved in 100 grammes of 80% alcohol. The solution must be kept in a dark colored, well stoppered bottle as the solution soon decomposes when exposed to the light. The original Günzberg formula was composed of the same amount of the ingredients dissolved in 30 c.c. of absolute alcohol. This solution still more readily undergoes decomposition and has no advantages over the above modification. The solution is employed in the following manner; four or five drops of the reagent are mixed on a small porcelain dish, or small butter plate with an equal amount of the filtered gastric juice or the unfiltered gastric contents. This is placed on a water bath, kept just below the boiling point, and evaporated slowly. If free hydrochloric acid be present in the proportion of 0.5 p. m. or more a fine rose tint will develop at the edge of the drop where the mixture is dried.

The mixture may be evaporated over a naked flame with the same results provided the temperature is not raised above the boiling point. If too much heat is applied, a brown or brownish red color may develop, which resembles the color produced where free hydrochloric acid is absent. The rose color produced by this reagent only comes from free mineral acids; organic acids, acid salts, combined hydrochloric acid, peptone and albumose produce only a brown or yellowish discoloration.

THE RESORCIN TEST.

The solution consists of five grms. of resorcin [resublimed] and three grms. of cane sugar dissolved in 100 c.c. of 94% alcohol. Six drops of the filtered gastric juice and three drops of the solution are mixed on a porcelain plate and slowly evaporated as in the phloroglucin-vanillin [Günzberg] test. Care must be employed that too much heat is not applied, as heating too strongly simply yields a brown or black deposit. If the operations be properly conducted and free hydrochloric acid be present a fine vermilion

red line forms at the edge of the drops, following down the edge of the solution as evaporation proceeds, while the color at the periphery gradually fades, disappearing entirely after a short time, leaving a reddish brown stain. This test has the same degree of delicacy as the phloroglucin-vanillin test and the advantage of much greater stability, retaining its delicacy for months while the latter lasts only a few weeks.

Many other tests might be mentioned some of them much less delicate, among them tropaeolin OO Mohr's reagent, methyl violet, and emerald green, but the three described will be found the most reliable and easily applied.

COMBINED HYDROCHLORIC ACID.

If albuminous bodies are treated with a weak solution of hydrochloric acid it is found that a certain amount of the hydrochloric acid combines with the albuminous bodies to form compounds which do not give the reactions of free hydrochloric acid. In other words certain affinities of the albuminous substance must be saturated before hydrochloric acid appears in the free state. In the stomach the same reaction must take place except probably to a greater extent due to the more complicated chemical processes through which these substances pass. This is shown by the fact that even after a simple test meal a certain amount of time elapses before the presence of free hydrochloric acid can be demonstrated. In the Ewald meal from 30 to 40 minutes elapses before free hydrochloric acid can be demonstrated in the normal individual while in the more complex meals, considerably more time is required. This form of hydrochloric acid is important in as much as it constitutes a part of the physiological hydrochloric acid and stomach digestion will proceed in a fairly normal manner if enough hydrochloric acid is secreted to saturate these affinities while not enough is secreted to form the excess or reserve supply called free hydrochloric acid. It is evident therefore that if free hydrochloric acid is present all these affinities must be saturated, while in the absence of free hydrochloric acid, some hydrochloric acid, enough to more or less saturate these affinities may have been secreted. The entire absence of hydrochloric acid both free and combined if more than temporary, is a serious condition, indicating an atrophy of the gastric mucosa

or a severe gastric catarrh. From these considerations it will be seen how important the determination of the combined hydrochloric acid is, in all conditions of anacidity. The estimation and quantitative determination of the combined hydrochloric acid will be deferred to the paragraphs devoted to the quantitative determination of hydrochloric acid.

The following table* shows the amount of pure hydrochloric acid necessary to combine with 100 grammes (or 100 c.c.) of the various food stuffs.

Milk.....	0.32 to 0.42 gramme	of pure HCL.	
Sweetbread (boiled).....	0.9	"	"
Calve's brain (boiled).....	0.65	"	"
Liver sausage.....	0.8	"	"
Mettwurst.....	1.0	"	"
Blood sausage.....	0.3	"	"
Graham bread.....	0.3	"	"
Pumpernickel.....	0.7	"	"
Wheat bread.....	0.3	"	"
Rye bread.....	0.5	"	"
Beer (German).....	0.07 to 0.15	"	"
Beef (boiled).....	2.0 grammes	"	"
Mutton (boiled).....	1.9	"	"
Veal (boiled).....	2.2	"	"
Pork (boiled).....	1.6	"	"
Ham (raw).....	1.9	"	"
Ham (boiled).....	1.8	"	"
Cervelat sausage.....	1.1	"	"
Swiss cheese.....	2.6	"	"
Fromage de Brie.....	1.3	"	"
Edam cheese.....	1.4	"	"
Roquefort cheese.....	2.1	"	"

LACTIC ACID; FORMATION, SIGNIFICANCE, DETECTION.

It was formerly supposed that lactic acid was secreted by the stomach, but by the more accurate investigations of later years, it has been shown beyond doubt that lactic acid in the gastric contents is either introduced as such in the food or is the product of abnormal fermentative changes in the food after ingestion.

Lactic acid may be introduced in food either as sarcolactic acid

*Erich:—Dissert Erlangen, 1893.

from meat or fermentation lactic acid found in bread and other starchy foods. Lactic acid may be formed after the food is ingested, in cases of carcinoma of the stomach, and probably also in small amounts in other conditions, of subacidity or anacidity associated with deficient motility.

In the great majority of cases of carcinoma of the stomach lactic acid is present in considerable amounts, except in those cases in which the motility is not impaired. In such cases only a small amount of lactic acid can be demonstrated usually and in some cases its presence can not be demonstrated. There are cases of carcinoma of the fundus or body of the stomach in which the motility is so good that at the end of one hour, no remains of the test meal can be regained.

Small traces of lactic acid can usually be detected for some time after the administration of the Ewald breakfast or similar meals, though at the height of digestion the usual tests are negative, due either to the absorption of the lactic acid or the interference of free hydrochloric acid or of the products of digestion with the delicacy of the tests. In cases in which it is desirable to prove the formation of lactic acid within the stomach, it is necessary to employ some meal which is entirely free from lactic acid.

Such a meal has been proposed by Boas consisting of oatmeal gruel to which only a little salt has been added. The stomach is washed out on the evening preceding the administration of the meal until no food particles can be found, the gruel given in the morning and the contents removed one hour after.

Only rarely, under such conditions, is any notable amount of lactic acid to be demonstrated except in cases of carcinoma of the stomach. The easiest clinical test for the presence of lactic acid is that of Uffelman. Ten c.c. of a 4% solution of carbolic acid are mixed with twenty c.c. of water, and a drop of a strong solution of ferric chloride added. A beautiful amethyst blue color is produced which turns a canary yellow when treated with a solution of lactic acid or gastric juice containing lactic acid. The delicacy of this test is interfered with by the presence of free hydrochloric acid and by presence of peptones. Glucose, acid phosphates and alcohol give a reaction resembling that of lactic acid, butyric acid giving a much lighter tint. In case of doubt, a modification that has given good

results is the following: Five or ten c.c. of the filtered gastric juice are treated with ten times its volume of ether free from alcohol and then shaken in a stoppered separating funnel for fifteen or twenty minutes and allowed to stand till the layers have separated. The ethereal solution is allowed to evaporate, the residue dissolved in five or ten c.c. of water, and the solution tested for lactic acid in the same manner. While this test is not a very delicate one, lactic acid when present in considerable amounts gives a more decided reaction than any of the substances mentioned as having a similar reaction, and is a good test for clinical purposes.

Boas' method is to be employed in doubtful cases. This method is based upon the fact that when lactic acid is treated with strong oxidizing agents, formic acid and acetic aldehyde are formed.



Acetic aldehyde may be easily recognized by its action on Nessler's reagent, or upon an alkaline solution of iodine in iodide of potassium. Nessler's reagent is prepared in the following manner.

One hundred c.c. of a 4% solution of iodide of potassium is warmed and while warm treated with iodide of mercury until a small amount remains undissolved. After cooling forty c.c. of water are added. Two parts of this solution are then treated with three parts of a strong solution of caustic potash, any precipitate which may form is filtered off and the reagent kept in a well stoppered bottle.

The solution of iodine is prepared by mixing a solution of iodine in iodide of potassium with caustic potash or potassium carbonate.

Method: The filtered gastric juice is tested for the presence of free acids as above, and if present ten or twenty c.c. are treated with an excess of barium carbonate. If no free acids are present this is not necessary. The solution is now evaporated to a syrup on the water bath to drive off the fatty acids. The syrup is treated with a few drops of phosphoric acid and brought to a boiling point to expel carbon dioxide. After cooling it is extracted with one hundred c.c. of ether by shaking for half an hour. After standing for a short time to allow separation to take place, the ethereal layer is drawn off and evaporated [avoiding a flame] the residue taken

up in forty-five c.c. of water, shaken and filtered. The filtrate is treated in an Erlenmeyer flask with five c.c. of strong sulphuric acid and as much black oxide of manganese as will lie on the point of a knife blade. The flask is closed with a perforated stopper, in which is placed a bent glass tube, the long arm passing into a cylinder filled with ten or fifteen c.c. of the Nessler's reagent or alkaline iodine solution prepared as described. Carefully heat the flask and if lactic acid is present, aldehyde will distill over forming aldehyde mercury, yellowish red in color, if Nessler's reagent is used and yellowish crystals of iodoform, which may be recognized by their odor, if the alkaline solution of iodine is employed.

1. *Butyric acid* can usually be determined by its odor alone which is that of rancid butter. In case of doubt, ten c.c. of the gastric juice are extracted with fifty c.c. of ether, the ethereal solution evaporated, the residue taken up with water. The odor is more evident in this concentrated aqueous solution. A small amount of calcium chloride causes the separation of an oily layer of butyric acid, strong mineral acids also separate the oily layer or drops of the acid.

Acetic acid may also be detected by its odor.

2. Ten c.c. of the gastric juice are extracted with ether, the ether evaporated, the residue taken up with a small amount of water, accurately neutralized with caustic soda solution and mixed with a few drops of a very dilute solution of ferric chloride. In the presence of acetic acid this gives a dark red color.

3. The ethereal residue after evaporation is taken up with a small amount of strong sulphuric acid and alcohol. If acetic acid is present the fragrant odor of ethyl acetate is easily detected.

Fatty acids do not occur normally in the stomach contents. Butyric acid may be formed when a large amount of milk or carbohydrates have been ingested, usually associated with an excess of lactic. It has been shown also that butyric acid can be formed from lactic acid.

Acetic acid on the contrary is a product of alcohol and may be formed from alcohol ingested or from alcohol produced by the action of yeast upon the sugar contained in the stomach contents. Hence it follows that it is necessary to exclude alcoholism before significance is attached to the presence of acetic acid in the stomach

contents. If in the case of acetic acid, alcoholism be excluded, and in the case of butyric acid the ingestion of butter or fats in general be excluded, the presence of these acids has the same significance as the occurrence of lactic acid viz, stenosis of the pylorus with dilatation and fermentation.

LECTURE XVII.

QUANTITATIVE ANALYSIS OF THE STOMACH ACIDS.

NUMEROUS methods have been devised for the estimation of the amount of free hydrochloric acid, present in the gastric juice.

The most convenient method of estimation for clinical purposes is that of Töpfer, which at the same time, estimates the acidity due to organic acids and acid salts, and that due to the combined hydrochloric acid.

Method: Three indicators are used in this method;

1. A 0.5% alcoholic solution of dimethyl-amido-azo-benzol.
 2. A 1% aqueous solution of alizarin.
 3. A 1% alcoholic solution of phenol phthalin.
1. As has been mentioned under the head of tests for free hydrochloric acid, dimethyl-amido-azo-benzol reacts to very faint traces of mineral acids particularly hydrochloric, reacts to organic acids only when present in very large amounts, and not at all to combined hydrochloric acid or acid salts. It will be seen that by this indicator we can easily find the amount of free hydrochloric acid.

Ten c.c. of the filtered gastric juice are measured into a small clean flask and a few drops of dimethyl—amido—azo—benzol added. The solution turns a bright red in the presence of free hydrochloric acid. The solution is now titrated with a decinormal solution of caustic soda, (prepared as above) until the red color of the solution changes to a clear yellow.

2. Into a second beaker or flask, ten c.c. of the gastric juice are measured, a few drops of the alizarine solution added and the solution titrated with the decinormal solution of caustic soda until the solution turns to a clear violet color.

As this tint is difficult for the unpracticed eye to recognize, Töpfer recommends the following preliminary tests.

(a) To five c.c. of distilled water, add two or three drops of the alizarine solution. A clear yellow color results.

(b) To five c.c. of a one per cent solution of disodium phosphate add the same amount of the alizarine solution.

A reddish color with a slight tinge of violet results.

(c) Five c.c. of a one per cent solution of sodium carbonate when treated with the same amount of alizarine give a clear violet tint, which is the tint to be reached in the titration. Until the eye becomes accustomed to the reaction it is well to prepare this solution as a guide in the titration.

3. To a third portion (ten c.c.) of the filtered gastric juice, two or three drops of phenol—phthalein solution are added and the solution titrated with the decinormal solution of caustic soda. After a certain amount of the solution, has been added, a light rose color develops, which is not however the end of the reaction. It will be noticed that as the drop of caustic soda solution falls into the solution, a dark red color is produced at the place of contact, fading into rose color on agitation. The titration must be carried on until the entire solution has reached this color and no line of separation can be made out on adding a drop of the caustic soda solution.

There are two ways of stating the result of the titrations: The simplest method is to state the number of c.c. of the caustic soda solution which would be necessary to neutralize one hundred c.c. of the gastric juice as that number of degrees of acidity. For example the number of c.c. of the caustic soda solution necessary to neu-

tralize ten c.c. of the gastric juice, using dimethyl—amido—azo—benzol as an indicator, is 2.3 c.c. One hundred c.c. would then require ten times that, the amount of acidity being stated as twenty-three degrees.

The second method of stating the results is to give the amount of acid per mille in terms of hydrochloric acid. As each c.c. of the solution of caustic soda will neutralize 0.00365 gramme of pure hydrochloric acid, the above example would show 0.8395 gramme of hydrochloric acid per mille or 0.8365 per cent.

As an example of the calculations employed in Töpfer's method, let us suppose that in the titration (1) with dimethyl—amido—azo benzol as an indicator, 3.5 c.c. of caustic soda solution were employed, (2) with alizarine 4.9 c.c. of the caustic soda solution were required and (3) with phenol phthalein 7.5 c.c. of caustic soda solution were required to produce the proper tint, using in each case ten c.c. of the stomach contents.

1. As dimethyl—amido—azo=benzol reacts only with free hydrochloric acid, the acidity referable to this is 35 degrees or .12775 per cent.

2. Alizarine shows the tint of an alkaline reaction when the free hydrochloric acid, organic acids and acid salts have been neutralized, combined hydrochloric acid having no effect upon it. Hence it follows that by subtracting the amount of free hydrochloric acid from the acidity found by alizarine, the amount of acidity due to organic acids and acid salts will be found, in this case $49 - 35 = 14$ degrees or .0511 per cent.

3. Phenol phthalein only turns to a dark red color when all the acidities of the solution have been saturated, including the combined hydrochloric acid. The amount of combined hydrochloric acid may be found by subtracting the acidity found by alizarine from that found by phenol phthaleine, in this case $75 - 49 = 26$ degrees or .0949 per cent.

METHOD OF MARTIUS AND LUTTKE:— By this method, the amount of physiological hydrochloric acid, the free and combined hydrochloric acid are found as well as the total chlorine of the gastric juice by determination of the amount of chlorine. The method is based upon the fact, that by moderate incineration, the free hydrochloric acid can be driven off, while the chlorine in combination

with the inorganic bases is not affected.

For this method the following solutions are required:

1. A decinormal solution of hydrochloric acid which can be prepared by standardizing against the decinormal caustic soda solution as described in a former chapter.

2. A decinormal solution of nitrate of silver, containing 25% of pure nitric acid. This solution is approximately made up by dissolving 17 grammes of pure crystallized nitrate of silver in 900 c.c. of a 25% solution of nitric acid and adding 50 c.c. of the liquor ferri sulphur oxydati of the German pharmacopœia (the liquor ferri oxysulphatis N.F.) will serve the same purpose. The solution is then standardized against the solution of hydrochloric acid and diluted to the proper volume. Each c.c. of the solution is equivalent to 0.00365 gramme of pure hydrochloric acid.

3. A decinormal solution of ammonium sulphocyanate. Eight grammes of the pure salt are dissolved in 900 c.c. of distilled water and titrated against the decinormal solution of silver nitrate. After ascertaining the strength of this solution it is diluted so that it is exactly equivalent to the decinormal solution of nitrate of silver.

METHOD:—1. To determine the total amount of chlorine present in the gastric juice, ten c.c. of the stomach contents after thorough mixing, are measured into a small cylinder graduated to 100 c.c. and treated with twenty c.c. of the solution of nitrate of silver. The mixture is thoroughly shaken and allowed to stand for ten minutes. The mixture is then diluted to 100 c.c. once more agitated, and filtered through a dry filter into a dry flask. Fifty c.c. of the filtrate are then titrated with the decinormal solution of ammonium sulphocyanate until a permanent red color appears. Multiply the number of c.c. of ammonium sulphocyanate by two as only half the filtrate was taken and subtract from the number of c.c. of nitrate of silver added, (20) the result will be the number of c.c. of the nitrate of silver solution precipitated by the total chlorine of the gastric juice and correspond to the same number of c.c. of decinormal solution of hydrochloric acid, the whole amount of chlorine being expressed in terms of hydrochloric acid.

2. To determine the amount of chlorine in combination with inorganic bases.

Ten c.c. of the filtered gastric juice or of the well mixed sto-

mach contents are evaporated to dryness in a platinum or porcelain crucible over a water bath or on a plate of asbestos to avoid loss from sputtering. The incineration is carried only to the point when the residue ceases to burn with a luminous flame. After cooling the residue is treated with distilled water up to about 100 c.c. or until the filtrate comes away free from chlorides which may be shown by treating a drop of silver nitrate. If the filtrate remains perfectly clear after the addition of a drop of nitrate of silver the residue is free from chlorides. To the clear filtrate is now added ten c.c. of the decinormal solution of nitrate of silver and the excess titrated by means of the decinormal solution of ammonium sulphocyanate as before. The amount of ammonium sulphocyanate solution subtracted from the amount of the silver solution (ten c.c.) gives the amount of silver precipitated by the chlorides remaining after incineration, in combination with the inorganic bases. By subtracting the result of the second process from that of the first, the amount of free hydrochloric acid and of combined hydrochloric acid is determined.

MODIFICATIONS:— 1. By titrating with decinormal caustic soda solution using dimethyl—amido—azo—benzol as an indicator we obtain the amount of free hydrochloric acid, this subtracted from the combined amount of free and combined hydrochloric acid, will give the amount of combined hydrochloric acid.

2. By determining the total acidity with phenol phthalein and subtracting from it the amount of free and combined hydrochloric acid we can estimate the acidity due to organic acids and acid salts.

3. The amount of organic acid present may be estimated in terms of hydrochloric acid by the method of Hehner—Seeman (to be described later.) This result deducted from the result of the preceding (No.2.) test gives the amount of acidity due to acid salts.

LEO'S METHOD: Leo bases his method upon the fact that when calcium carbonate is added in a fine powder to the gastric juice, the free and combined hydrochloric acid combine with the calcium carbonate to form calcium chloride, a neutral salt, while the acid salts are not affected. During the reaction however the calcium chloride reacts with the phosphates to form acid calcium phosphate, (mono-calcium phosphate, CaHPO_4 .) As this requires double the amount

of caustic soda solution to neutralize that would be required for the acid sodium phosphate, it is necessary to add each time an excess of calcium chloride solution before titration.

METHOD: Ten c.c. of the gastric juice are shaken up with fifty c.c. of ether to remove organic acids. The residue after drawing off the ethereal layer is treated with five c.c. of a concentrated solution of calcium chloride and titrated with the decinormal solution of caustic soda using phenol phthalein as an indicator. This determines the acidity due to free and combined hydrochloric acid and to acid salts. A second portion of fifteen c.c. is treated with a small amount of pure dry calcium carbonate the mixture stirred and immediately filtered through a dry filter. The carbon dioxide is expelled from the filtrate by passing a current of air through it. Ten c.c. of the filtrate are then treated with five c.c. of the saturated solution of calcium chloride and titrated as above. The acidity found is due to the acid phosphates. By subtracting the result found in the second titration from that of the first, the amount of free and combined hydrochloric acid is determined.

BOAS' METHOD: This method is an easily applied test for free hydrochloric acid which gives fairly accurate results in the absence of organic acids or when they are present only in traces. Ten c.c. of the filtered gastric juice are titrated with decinormal caustic soda solution until a small amount (a drop) removed by a glass rod, fails to change the tint of congo paper. Instead of using the paper as an indicator outside, a small bit of the congo paper may be dropped into the solution and the titration conducted slowly, with constant shaking until the paper regains its original red color. This test however can not be employed in the presence of any considerable amount of free organic acids.

LACTIC ACID — QUANTITATIVE ESTIMATION.

A simple clinical test for lactic acid has been devised by Strauss. A separating funnel is graduated to five c.c. below and twenty-five c.c. above. The funnel is filled to the five c.c. mark with gastric juice and ether added to the twenty-five c.c. mark. The funnel is corked and well shaken and after standing for a short time to allow the fluids to separate, the liquids are run out to the five c.c. mark. Distilled water is added to the twenty-five c.c. mark

and the mixture treated with two drops of a solution of the official tincture of the chloride of iron diluted 1:10. On shaking the mixture, an intense green color is produced if lactic is present in the proportion of 1 p. m. or more. If present in the proportion of from 0.5 to 1 p. m. only a pale green color is produced.

BOAS' METHOD:

This method of estimating the amount of lactic acid depends upon the oxidation of lactic acid into aldehyde and the estimation of aldehyde by means of a standard solution of iodine.

SOLUTIONS REQUIRED:

1. A decinormal solution of iodine is prepared by dissolving twenty-five grammes of potassium iodide in about two hundred c. c. of water, and dissolving in this 12.6 grammes of resublimed iodine. The solution is diluted with distilled water to 1000 c.c. and requires no correction.

2. A decinormal solution of sodium arsenite. Dissolve 16.5 grammes of sodium arsenite in about 900 c.c. of distilled water. It is then titrated against the decinormal solution of iodine and diluted so that the two solutions are equivalent.

3. Hydrochloric acid (Sp.Gr. 1018.)

4. Normal solution of potassium hydrate (56 grammes in 1000 c.c.)

METHOD:

Ten or twenty c.c. of the filtered gastric juice are tested for the presence of free acid, if present a small amount of barium carbonate is added, if free acid is absent this addition unnecessary, and evaporated to a syrup. A few drops of phosphoric acid are added and the solution boiled slightly to expel carbon dioxide.

Allow the syrup to cool, extract with 100 c.c. of ether free from alcohol, after the two fluids have separated draw off the ethereal solution, evaporate, take up the residue in 45 c.c. of water, filter. The filtrate is treated in an Erlenmeyer flask with five c.c. of sulphuric acid and a small amount of manganese dioxide. The flask is closed by a two holed rubber stopper, one aperture being closed by a glass tube and rubber tubing clamped off, the other opening receiving a bent glass tube leading to the distilling apparatus. The distillate is received in a large flask well stoppered. The mixture is distilled at a gentle heat until about four fifths of the fluid has passed over.

The distillate is then treated with twenty c. c. of the decinormal solution of iodine and the same amount of twenty c.c. the normal potassium hydrate solution, thoroughly shaken and allowed to stand for a few minutes in the flask. Twenty c.c. of hydrochloric acid and an excess of sodium bicarbonate in powder are then added and the excess of iodine determined by titration with the solution of sodium arsenite. The sodium arsenite is added until the solution is decolorized, fresh starch solution added and the iodine solution added until the blue color becomes permanent. Each c.c. of the iodine solution in excess of the sodium arsenite solution is equivalent to 0.003388 gramme of lactic acid.

FATTY ACIDS:

The Method of Cahn and Mehring modified by McNaught is simple and fairly accurate. The total acidity is determined in ten c.c. of the filtered gastric juice by titrating with a decinormal solution of caustic soda using phenol phthalein as an indicator. Ten c.c. are evaporated to a syrup on the water bath, made up to the original volume with distilled water and the acidity determined as before. The difference in acidity will be the amount due to fatty acids.

TOTAL ORGANIC ACIDS:

The total organic acids are best estimated by the method of Hehner—Seeman, called by Leube, Brauns method.

Ten c.c. of the gastric juice are accurately neutralized with a decinormal solution of caustic soda using phenol phthalein as an indicator. This solution is then evaporated to dryness, carefully avoiding sputtering, and incinerated as long as the residue burns with a luminous flame. After cooling, the residue is extracted with boiling distilled water, filtered, and the amount of sodium carbonate formed determined by titration with a decinormal solution of hydrochloric acid. As the presence of free carbon dioxide interferes somewhat with the delicacy of the reaction when phenol phthalein is used as an indicator, the following modification has given better results. After the incinerated mass has been extracted with boiling water and filtered, a known excess of the decinormal solution of hydrochloric acid is added, the solution boiled to expel any carbon dioxide in solution, and the excess of acid determined by back titration with a decinormal solution of caustic soda.

This method is based upon the fact that when salts of the organic acids with the alkalies are incinerated at a low heat, the carbonates of the alkalies are formed with the liberation of water and carbon dioxide.

LECTURE XVIII.

DIGESTIVE FERMENTS — PRODUCTS OF DIGESTION — TESTS FOR SAME.

SALIVA. The saliva as found in the mouth is the mixed secretions of all the salivary glands. It may be readily obtained for testing by requesting the individual under examination to chew a piece of soft rubber or other insoluble substance to stimulate the secretion and as it forms it is placed in a clean receptacle. It is a clear, slightly opalescent fluid, of a slimy consistency, having a specific gravity of 1002 to 1006. Under normal conditions it has a slight alkaline reaction, its alkalinity averaging in man 0.08 per cent expressed as sodium carbonate (Chittenden.)

Its active constituent, ptyalin, acts most readily upon boiled starch, raw starch being protected from its action by the coating of cellulose surrounding each granule. Its action is entirely amylolytic, as it has no action upon other food products.

Its action upon starch may be demonstrated in the following simple manner. A few c.c. of boiled starch paste are treated in a test-tube with a few drops of saliva. A few drops removed and treated on a testing plate with a few drops of iodine solution give

the characteristic blue color of starch. After a moment or two a few drops removed will show a violet color, and by treating a portion at intervals the color changes gradually to a deep reddish brown and finally disappears. Different products of the action of the ferment are found at different stages of digestion. The violet color first found is a color which results from a mixture of erythrodextrin and starch when treated with iodine. Later the color becomes reddish brown due to the change of the starch entirely into dextrins and sugar. When digestion has gone on until the solution gives no color whatever with iodine, the solution still contains some form of dextrin (achroödextrin) as may be shown by the addition of alcohol which throws down a profuse white precipitate. It may be shown also that the solution contains sugar by treating a small amount of the mixture with Fehling's solution. This sugar according to the investigations of Nasse, v. Mering and Musculus is not dextrose as formerly taught but maltose.

The action of ptyalin is most energetic at the temperature of the body. It acts best in a neutral medium though a small trace of alkali has little or no effect upon it. Its activity is stimulated by the addition of enough acid to combine with its proteid constituents. A minute trace of acid still allows the action to continue, but for practical purposes we may say that the addition of free acids in such quantity as are found in the gastric juice, not only stop its action but entirely destroy the ferment so that after neutralization, it is no longer able to digest starch.

In the stomach the action of the ptyalin probably continues until the presence of free acid destroys the ferment. As no free acid can be demonstrated in the gastric until the lapse of fifteen or twenty minutes normally, the greater portion of the starch is transformed into sugar and achroödextrin. Under normal conditions then we should find in the gastric juice removed for examination, sugar, achroödextrin, and a faint trace of erythrodextrin. The presence of a marked reaction of erythrodextrin then is valuable presumptive evidence of hyperacidity, its absence indicating either normal, acidity or subacidity.

Only in rare cases have cases of the absence of ptyalin from the saliva been seen.

There are some unexplained cases in which with a normal or

diminished acidity, even the digestion of starches is very poor, as is shown by the marked reaction of erythrodextrin and the small percentage of sugar found by quantitative test. The activity of the salivary excretion ought always to be examined in such cases.

PESPIN.

The proteolytic ferment of the gastric juice is active only in an acid medium and is destroyed by very dilute solutions of the alkali carbonates. Pepsin is probably not secreted as such, its precursor being pepsinogen or propepsin which is transformed by weak acids into the active ferment, pepsin. While hydrochloric acid acts best in thus transforming pepsinogen into pepsin, other acids to a lesser degree perform the office. Pepsin, like the other ferments has the property of changing an almost unlimited amount of proteids providing the products of its action are removed when formed, and the temperature kept at a favorable point, as it appears to act by its presence, not being itself destroyed or changed by the reaction.

While no quantitative methods have been devised, and it has never been isolated in a pure state we know that a product can be obtained by complex chemical methods, which, while intensely proteolytic, exhibits none of the reactions of exproteids, so that the ferment whatever its nature probably is not a proteid.

The only tests which can be used for its detection therefore are of a qualitative nature, its effect in acid solution on proteid substances. Comparative tests may also be instituted, using for comparison, gastric juice from a healthy stomach.

The amount of acid necessary for the most vigorous action of pepsin varies with the form of proteids employed. For example, pepsin acts best on fibrin when the acidity is about one per thousand, while coagulated egg albumen is digested most rapidly when the acidity amounts to two or three per thousand of hydrochloric acid.

TEST.

Three test tubes or small wine glasses are taken and a small thin slice of boiled egg albumen placed in each. To the first is added three c.c. of the gastric juice; to the second, three c.c. of the gastric juice to which hydrochloric acid has been added in sufficient quantity to bring the acidity to two or three per thousand; the

third is acidulated as in number two and a few grains of pepsin added. The three tubes or glasses are now placed in the warm oven at a temperature of 40°C and allowed to remain for three hours.

If at the end of this time all three tubes show digestion by the rounding off and solution of the egg albumen the specimen contained pepsin, if number two and three only, show digestion the contents contained pepsinogen but no pepsin, while if only the third tube or glass shows traces of digestion the specimen contained neither pepsin nor pepsinogen.

PEPSINOGEN.

This substance is supposed to be excreted by the cells of the gastric mucosa, and to be changed into pepsin by the action of the hydrochloric acid of the gastric juice. This action has been differently explained by various experimenters, the most plausible theory being that a combination of the two takes place, with the formation of pepsin hydrochloric acid.

In the absence of hydrochloric acid, this body, pepsinogen, may be present in normal amount and require only the addition of a sufficient quantity of hydrochloric acid to bring the gastric juice to a normal acidity, to render the stomach contents active.

In the absence of free hydrochloric acid, we may test for the presence of this substance by acidulating with hydrochloric acid as in number two of the pepsin test, adding a small bit of boiled egg albumen and placing in an oven at a temperature of 40°C. for three hours, at the end of this time noting the presence or absence of signs of digestion.

Boas employs a comparative test which in doubtful cases may yield valuable information. Properly labeled tubes are prepared, and in them are placed measured quantities of gastric juice diluted with a solution of hydrochloric acid of the normal strength of the gastric juice (two or three p. m.) so that the tubes contain the gastric juice in dilutions of 1 : 10, 1 : 20. To each tube a small flake of egg white or fibrin is added and put in a warm oven at the temperature of the body. From the amount of dilution at which digestion ceases an idea may be gained of the amount of pepsin or pepsinogen which any gastric juice contains. For comparison, similar tubes may be prepared of normal gastric juice, and the digestive power of the two compared.

RENNET AND RENNET ZYMOGEN.

In addition to pepsin the gastric juice also contains a ferment or its zymogen, whose special property appears to be the precipitation of casein from milk. As in the transformation of pepsinogen into pepsin, hydrochloric acid is required, so rennet zymogen, in the gastric juice, is not transformed into rennet except in the presence of hydrochloric acid. Certain neutral salts of lime, such as calcium chloride have however the power of transforming rennet zymogen into rennet even in neutral or slightly alkaline solution.

The following tests for the presence of rennet and its zymogen have been devised by Boas.

RENNET.

Five c. c. of the gastric juice are exactly neutralized with a decinormal solution of caustic soda, five c.c. of neutral milk added, and the mixture after being well shaken is placed in an incubator at the body temperature.

If rennet is present the casein will form a firm coagulum in from ten to fifteen minutes.

A relative quantitative estimation of the rennet ferment may be performed by the following method:

The gastric juice is accurately neutralized and portions of this diluted with distilled water, in known proportions 1 : 10, 1 : 20 etc. To five c.c. of each of these dilutions, five c.c. of neutral milk are added and the tubes placed in the thermostat at the body temperature for fifteen minutes. At the end of this time the tubes are removed and the dilution at which no coagulation takes place is noted. In stating the dilution note must be taken of the fluid added in neutralizing.

RENNET ZYMOGEN.

Five c.c. of the gastric juice are rendered faintly alkaline by the addition of a decinormal solution of caustic soda, one c. c. of a one percent solution of calcium chloride and five c.c. of neutral milk are added. The tube is placed in the thermostat and after fifteen minutes should show a firm cake of casein.

QUANTITATIVE.

The gastric juice is rendered faintly alkaline by adding a decinormal solution of caustic soda and dilutions prepared, 1 : 10, 1 : 20 etc., estimating in the dilution the amount of fluid added in alka-

linizing. Five c.c. of each of these dilutions are placed in test tubes with five c.c. of neutral milk and one c.c. of a one per cent solution of calcium chloride. These are placed in a thermostat at the body temperature and at the expiration of fifteen minutes the dilution at which the enzyme fails to act is noted. From the observations of Boas and others it appears that the secretion of the ferments and the proenzymes is less affected by the minor disturbances which may cause a temporary arrest of the acid secretion of the stomach. Decrease in the activity of the ferments on the other hand usually is the result of some organic change in the gastric mucosa.

By experiment upon normal individuals it has been found that rennet is active in dilutions of from 1:30 to 1:40 and rennet zymogen in dilutions varying from 1:100 to 1:150. It has been found that even in the absence of free hydrochloric acid the ferments may be active up to the limit observed in normal individuals and that in such cases the condition of anacidity was a temporary matter, due to some mental or circulatory disturbance, the acid reappearing when the cause of the disturbance was removed.

On the other hand, in cases of anacidity in which the rennet zymogen was active only in the stronger dilutions 1 : 5, 1 : 10 etc., the anacidity is due to some organic change in the gastric mucosa from which recovery is usually rare.

It will be seen from these considerations, of what importance the quantitative investigation of the gastric ferments is from the prognostic standpoint.

ACTION OF PEPSIN ON PROTEIDS.

The action of pepsin upon proteids as has been shown, only takes place in an acid medium. The action is a very complex one and is not as yet fully understood. The first result seen of the action of an hydrochloric acid solution of pepsin upon a coagulated albumen such as egg-white is apparently a partially mechanical change. The egg white swells up, its edges become rounder and it becomes clearer and more glassy in appearance. The egg white then begins to dissolve as is shown by the presence in the solution of a substance precipitated by neutralization which may be called syntonin or acid albumen. This action takes place also in acid solutions to which the pepsin has been added. The next step is one in which the pepsin plays an important part. The syntonin or acid

albumen is changed first into the primary albumoses proto- and hetero-albumose. These undergo further change and become deuto albumoses and finally peptones. These substances may be distinguished from each other by the following reactions.

Native albumens may be removed from the solution if present by rendering the stomach contents faintly acid if not already so and boiling. The precipitate will consist of the native proteids, viz, albumen and globulin.

(b) The solution is carefully neutralized by the addition of a weak caustic soda solution. The precipitate will consist of syntonin or acid albumen. The neutralization must be exact as the precipitate is dissolved by an excess of acid or alkali to form acid albumen or alkali albumen respectively.

(c) The filtrate from which the albumen and acid albumen has been removed is now saturated with magnesium sulphate and filtered. The precipitate which consists of the primary albumoses, proto and hetero albumose, is dissolved in water, placed in a dialyser and the salts removed by dialysis. As hetero-albumose is insoluble in pure water it is precipitated by the removal of the salts as in a dialyser. The proto-albumose remains in solution as it is soluble in water and may be tested for by acidulating with nitric acid in the cold, the precipitate redissolving on heating.

(d) Deuto albumose or secondary albumose is detected in the following manner; A sufficient quantity of the gastric juice is freed from albumen and acid albumen according to (a) and (b). The filtrate is saturated with powdered ammonium sulphate and the precipitate which forms, consisting both of primary and secondary albumoses is filtered off and the precipitate washed thoroughly with a saturated solution of ammonium sulphate.

The precipitate is redissolved in the least amount of water possible, faintly acidulated with acetic acid and saturated with common salt which precipitates the primary albumoses, leaving the deuto-albumose or secondary albumose in solution. After filtration the secondary albumose may be detected by saturating again with ammonium sulphate; any precipitate which may form consisting of deuto-albumose. It may be detected also by adding a considerable amount of common salt to its solution and acidulating with nitric acid. A precipitate will form in the presence of deuto-albumose

redissolved on heating.

(e) Peptone may be detected by precipitating all the other proteids by saturating with ammonium sulphate and filtering. The filtrate contains the peptone which may be tested for by the biuret reaction. The filtrate is treated with an excess of caustic alkali and few drops of a very dilute solution of copper sulphate. If peptones are present in the solution, a pink or rose red color appears.

PART SECOND.

The Gastric Clinic.

ACUTE GASTRITIS.



Simple Acute Gastritis, Phlegmonous or Purulent Gastritis, Suppurative Inflammation of the Gastric Mucosa, Abscess of the Stomach, Infectious Gastritis, Gastritis Mycotica or Parasitiasis, Gastritis Diphtherica and Crouposa, Toxic Gastritis, Gastritis Venenata.

CHRONIC GASTRITIS.

GASTRIC ULCER.

GASTRIC CARCINOMA.

ULCUS CARCINOMATOSUM.

MOTOR INSUFFICIENCY OR DILATATION.

GASTROPTOSIS.

ACUTE GASTRITIS.

1. Simple Acute Gastritis.
2. Phlegmonous or Purulent Gastritis—Suppurative Inflammation of the Gastric Mucosa — Abscess of the Stomach.
3. Infectious Gastritis — Gastritis Mycotica or Parasitiasia — Gastritis Diphtherica and Crouposa.
4. Toxic Gastritis — Gastritis Venenata.

GASTRITIS is a collective or generic term which comprehends all inflammatory processes proper of the stomach, including the so-called catarrh of the superficial layer of columnar epithelium, the inflammation of the glandular parenchyma and interstitial connective tissue, the purulent infiltration of the submucosa and, muscularis, and also the penetrating excoriations of corrosive poisons.

It is natural that these manifold morbid conditions should present considerable variations in etiology as well as in the intensity of the symptoms. It is almost impossible to draw a sharp limit separating the simple superficial catarrhs from the deeper, penetrating inflammations. Penzoldt suggests the line between mucosa and submucosa.

We may designate, as simple Gastritis that inflammation of the gastric mucosa which involves not only the superficial columnar epithelium, but as a rule the glandular parenchyma. This con-

dition may occur in an acute and in a chronic form and under each classification the acute and the chronic gastritis, one may arrange two subdivisions; (1) the primary and (2) the secondary gastritis.

We therefore have (1) the acute simple primary gastritis which occurs as the original disease, and (2) the acute secondary gastritis known as the gastritis sympathica acuta which occurs not as the original disease, but as a frequent accompaniment of numerous acute febrile disorders. All the exanthematous infectious diseases, measles, scarlatina, variola, typhus and typhoid fevers, puerperal fever, pyaemia, dysentery, croup and diphtheria are known to effect pathological changes in the gastric mucosa directly or to influence it detrimentally by reflex nervous action (Hoppe Seyler *Allgemeine Biologie*, 1877, p. 242.)

There is a very plausible desire evident in some recent works on the subject to avoid the name *Stomach or gastric catarrh*, because the word *catarrh* has reference to a superficial inflammation but in gastritis we are dealing also with parenchymatous inflammation. Penzoldt uses the expression simple gastritis for an inflammation reaching no deeper than the submucosa (*Gastritis simplex*) for the penetrating results of suppurative or purulent inflammation, he uses the term (*Gastritis gravis*) Grave gastritis. He does not favor the terms *toxic* and *infective* gastritis for to a certain extent all forms of this disease are toxic and infective and in his book (*Specielle Therapie Innerer Krankheiten*, Vol. iv, p. 320) he discusses only (1) simple and grave acute, secondly (2) chronic gastritis and thirdly [3] purulent or suppurative gastritis. Fleischer (*Specielle Therap. u. Pathol. d. Magen u. Darmkr. S. 793.*) describes (1) simple acute, (2) secondary or sympathetic acute, [3] phlegmonous or purulent [4] toxic [5] diphtheritic, croupous, mycotic, parasitic [6] chronic gastritis. Excepting those forms mentioned by Fleischer under group 5, Boas describes all of these in separate chapters.

Ewald mentions and describes all of these forms and subdivides the suppurative inflammation [the *Gastritis phlegmonosa purulenta*] into an idiopathic primary and a metastatic secondary form. Sidney Martin's treatise of the organic and functional diseases of the stomach deals with the symptomatology, pathology and treatment of acute and chronic gastritis in one chapter [the viii] and then goes on it to speak of toxic and infective gastritis in the next chap-

ter [the ix]. Albert Mathieu of Paris, briefly mentions acute and chronic gastritis and the varying amount of mucus and acid accompanying these diseases, none of the other forms are referred to. [Wm. Wood Edition, N. Y.]

Rosenheim, [*Pathol. u. Therap. d. Krankh. des Verdauungsapparates*, p. 99] describes gastritis acute, simple, phlegmonous, toxic, diphtheritic and chronic. Einhorn approaches the simple classification of Penzoldt and divides acute gastritis into [1] simple, [2] phlegmonous and [3] toxic and then proceeds to the consideration of chronic gastritis.

Alois Pick describes [1] acute, [2] infectious, [3] phlegmonous, (4) toxic, (5) parasitic and (6) chronic, (*Vorlesungen über Magen u. Darmkrankheiten*, S. 73.) and Fleiner (*Lehrbuch d. Krankheiten d. Verdauungsorgane*) gives an account of (1) gastritis catarrhalis acuta for which he also uses the name Gastricismus, [2] Gastritis toxica, [3] Interstitial suppurative gastritis, stomach abscess and stomach phlegmone, or gastritis phlegmonosa, or interstitialis, or submucosa purulenta, or also Linitis suppurativa, [4] Mycotic gastric inflammations, (5) Chronic gastritis.

Osler, in his new principles and practice of Medicine p. 348-359, consider; — Acute simple, (2) phlegmonous or acute suppurative, (3) toxic, (4) Diphtheritic or membranous, (5) Mycotic or parasitic, (6) chronic gastritis, under the latter he gives a special paragraph to the chronic forms with extreme connective tissue proliferation, increase in thickness of the submucosa and muscularis under the name of sclerotic gastritis.

These references are sufficient to demonstrate the discrepancy existing in latter works concerning the separate and distinct recognition of the various forms of this disease and that a more uniform classification would be desirable.

In accordance with Penzoldt, this treatise will describe only (1) simple acute gastritis, (2) simple chronic gastritis and separately, (3) the forms in which the element of pus formation is a factor, the suppurative gastric inflammations and in a supplement the forms due to toxic or corrosive agents and the remaining very rare varieties may be appropriately described.

One should be very careful not to diagnose every temporary, transient gastric disturbance as acute gastritis, nor a prolonged loss

of appetite, with eructations, coated tongue and no other demonstrable signs and symptoms as chronic gastritis, as Penzoldt correctly says this is in the majority of such cases, neither justifiable nor conducive to scientific development of diagnosis. We can agree with him in the opinion that it is inconceivable that all the functional and anatomical changes, which, one is accustomed to find in acute inflammations in other tissues, should really be present in every brief digestive disturbance after dietetic errors, alcoholic abuse, etc.

We could not designate a brief irritation of the nose with sneezing secretion of mucus and hoarseness, lasting several hours, as nasal catarrh. By catarrh of the air passages we understand a more lasting affection with a somewhat typical course and more permanent changes in the mucosa of both a structural and functional nature. Indeed Sidney Martin very appropriately considers these functional, lighter forms of gastric disturbance under a separate chapter and classifies them under (1) gastric irritation, (2) gastric insufficiency. Functional disorders, then are irregularities of gastric motility, absorption and secretion, and also of the innervation and vascular supply, in which organic disease of the organ—ulcer, gastritis, neoplasm, etc., are absent. It can not with certainty be stated that all histological changes are absent in functional disorders, at least in functional disorders of secretion. I have become convinced of certain changes in the acid and ferment cells that are apparently quite constant.

Ever since Beaumont's pioneer observations it has been known that every severe inflammatory irritation of the gastric mucosa produces an alteration in the gastric secretion, the quantity and effectiveness of which is much reduced; it is known furthermore that the impairment of one function of the stomach as a rule, rapidly involves that of another. The inner lining of the stomach can not in its true anatomical meaning of the word be called a mucus membrane, because it is devoid of one of the essential attributes of a mucus membrane, the mucus glands. The mucus of a normal stomach is surprisingly small in amount and owes its origin not to glands but to mucoid degeneration of the superficial columnar epithelial cells.

As this cylindrical epithelium continues down into to the al-

veoli of the peptic tubules without any distinct border line, all irritants striking the former must of necessity affect the parietal or border cells as well as the chief cells of the gland duct. It is characteristic for the pathology of gastric digestion, that impairment of one important function or rather of one of the many physiological processes of which the digestive act is composed, soon creates sympathetic disturbance in the remaining functions so that the clinical picture of an acute or chronic gastritis is that of a combination of disturbances.

It is not established nor very essential, which function suffers first, but probably in most cases a derangement of secretion starts the morbid series and the remaining functions follow in the affection; For example, if by ingestion of food which is already in a state of fermentation an acute gastritis has been induced, the reduction in the amount of hydrochloric acid produces a hindrance not only in the normal chemistry of the stomach but resorption and motility are also very soon retarded. This pronounced subacidity has in its consequence an imperfect digestion of the proteids so that very small amounts of acid albumen and hemialbumose are detectable in the vomit and peptone is found in traces only. A further step then is, that these undigested proteids continue to remain in the stomach longer than with normal proteolysis. This means a much more prolonged burdening of the gastric walls, the stomach does not gain sufficient rest in which to prepare itself for the demands of the following meal, the distention by the weight of the food lasts longer.

On the other hand much more of the carbohydrates will in this subacidity be converted into soluble starch, maltose and dextröse, than with a normal secretion of hydrochloric acid. With the progressing stagnation and putrefaction of proteids, these products of starch inversion mean more ready food for bacteria, which are constantly introduced with the saliva, and finding in the moist and suitable temperature of the gastric contents, congenial conditions for their development, the danger of progressive decomposition is very great. The toxic products of this carbohydrate and proteid decomposition, are irritants to the mucosa and increase the already existing inflammation.

When this inflammation has reached a certain stage an inflam-

matory oedema of the muscular layers sets in, effectually destroying the, motility, and simultaneously as in most all serous and mucus inflammations an alkaline transudate exudes into the mucosa, neutralizing the last vestige of hydrochloric acid that may yet be secreted. Lactic, butyric and acetic acid are evolved from the fermenting carbohydrates and further on H_2CO_3 and H .

When this latter CO_2 gas and hydrogen begin to expand and the already impaired motility can not expel them by eructation, the stomach is still further distended. The normal hydrochloric acid not only acts as an antiseptic and anti fermentative, but as we know undoubtedly, brings about energetic peristalsis, which effects a through mixing of the ingesta and frequently repeated contact and friction with those portions of the secretory membrane whose glands produce the hydrochloric acid and ferments. This mixing and triturating peristalsis is at the same time a most essential stimulus to absorption and eventually effects the timely expulsion of the chyme into the duodenum.

With impaired motility therefore the food masses remain too long in one and the same place. An intimate contact of the ingesta with the membrane as is produced by healthy peristalsis is essential for normal stimulation to continued secretion; hence the secretion of the oxyntic and ferment cells, already damaged by inflammatory infiltration soon ceases entirely. Resorption is not only impaired by absence of intimate contact with ingesta, but by the fact that the epithelial surface is in the various forms of gastritis covered with a tough glassy mucus, epithelial detritus, sometimes pus. In addition to this, one must not overlook the element of the inflammatory changes on the rate, tonicity, quality and quantity of the circulation on all of the gastric functions.

The damaging effects of inflammation might be partly made up again by a healthy peristalsis, but as this is not present resorption and secretion suffer very much. The solution the resorption must be looked upon as an act of self protection as there are nothing but poisons to absorb in these attacks. There is fortunately no excessive formation of peptone as this is prevented by the subacidity. So it is evident that in an acute gastritis there are numerous concurrent deleterious elements and changes, which are essentially similar to those of most light and severe gastric inflammations.

The clinical picture is a very manifold one as in the individual cases one may observe first one then another function that is most seriously damaged. It is natural to observe exceptions from the rule, thus in prolonged in acidity we may find cases in which the motility is unimpaired which of course favors intestinal digestion by timely evacuation of the chyme so that even the symptoms of dyspepsia may be lacking.

ETIOLOGY.

In the majority of cases, acute simple gastritis is caused by errors of diet. Irritation may be caused by quantity as well as quality of the food. Decomposed articles of liquid or solid nature will set up inflammation through the bacteria they contain. These germs must not be thought to invade the mucosa proper they exert effects by their action. Ewald (*l.c.*) says he has never found bacteria in the gastric tissues in these cases. Spoiled or decomposed meat, fish or vegetables, cheese, wine, cider or beer that has not completed its fermentation, infected milk, impure *pond* water have been known to produce severe acute gastritis.

EXCESSIVE INDULGENCE in perfectly healthy food can provoke the trouble, not only by the mechanical distention and irritation which is caused thereby, but by the inability of the motor power to move the ingesta about and to expel them into the duodenum and also by the deficiency in the secretion of gastric juice which may be able to digest a normal but not an excessive amount of food. The amount that can be digested under normal conditions without causing gastricismus will naturally vary considerably in different individuals.

CHEMICAL CAUSES. Among these may be mentioned quinine salts in large doses, all metallic salts, particularly those of copper, antimony, arsenic, lead, gold and silver; acids and alkalies unless properly diluted.

I have observed an acute gastritis follow the use of two grms. of sodium salicylate 3 times daily and feel convinced that iodide of potassium if not given properly mixed with food (right after meals) may lead up to gastritis. The various drugs used for gonorrhoea internally. Cubebs, copaiba and the oil of santalwood may in susceptible individuals bring about after long use a condition of the gastric mucosa in which acute gastritis is readily set up.

PSYCHIC CAUSES. It is said that grief, sorrow, terror, anger and even joy have been observed to produce acute gastritis. Sexual excesses particularly in neuasthenics are on record as causes.

THERMIC CAUSES. Large quantities of very cold or very hot liquids can produce the disease particularly when taken in rapidly when the body is in an overheated state.

MECHANICAL CAUSES. It is possible that pieces of fish bone, egg shells, oyster shell, fruit seeds if accidentally ingested may by mechanically scratching and bruising the mucosa cause a gastritis. I had occasion to observe a singular case of this disease in a professional base ball player by a blow from a base ball pitched with great speed. The bruise extended from the xyphoid cartilage to the left hypochondriac region. The player was knocked senseless and after partial recovery vomited a meal which he had taken two hours before mixed with blood and much mucus, later on he vomited some milk that was given him and on being tested this vomit was alkaline.

The pain was so severe that morphine had to be injected hypodermically and food was kept out of his stomach altogether for 3 days during which period he was fed by Boas nutrient enemata. The attack lasted two weeks and patient made a perfect recovery.

PREDISPOSITION. Manassein has shown that fever produced experimentally in dogs whom he had made anaemic by depriving them of much blood caused considerable suppression of the secretion of hydrochloric acid. And Kussmaul, Uffelmann, Leube and v. d. Velden have confirmed this subacidity in cases of fever in the human being. It is therefore what we should expect to find if we detect developing gastritis in convalescents from severe diseases, also in tuberculous, cancerous and syphilitic patients. Functional gastric disturbances predispose to acute gastritis as well as pre-existing or concomitant diseases of the heart, lungs, liver and kidneys. Ewald believes in hereditary predisposition to gastritis as some families show numerous cases of the trouble in spite of the best care they take of their stomachs.

IDIOSYNCRASY. It is a very perplexing fact that some person in good health acquire acute gastritis after certain articles of food. Whilst I was resident physician of Bay View Asylum, I had a collea-

gue, a perfectly robust vigorous man, who was not at all neurasthenic and who developed this disease every time he ate oysters. He could not be induced to eat them at any time after he established the causal relation, but convinced as by consenting to an experiment.

INFLUENCE OF SEX AND AGE. Acute gastritis occurs more frequently in men than in women; of 36 cases observed by myself of whom a record was taken, 10 occurred in females and 26 in males. Females, during menstruation and puerperium are more frequently attacked. Old persons and very young feeble children are more easily to be attacked than those in middle age. In nursing infants a very slight change in the milk may be sufficient to cause it. According to Booker of Baltimore, acute gastritis in infants is accompanied by prolongation of the time that the milk is retained in the stomach, at times over five hours, the gastric contents at times show epithelial and pus cells.

Rotch, (*Pediatrics*, p. 854.) holds that the acute form is more common in infants and that the chronic form while it does occur in them is more frequent in children toward puberty. The frequent attack of gastritis occurring during the hot summer months are undoubtedly largely due to the consumption of unripe fruit, Bouveret, however (*Traité des maladies de l'estomac*, p. 384, Paris, 1893.) attributes them to the abusive consumption of water. According to Pick the disease has been observed to develop after taking cold.

The effect of fever on the secretions of the stomach is not always present. Edinger, in five cases of fever found the secretion of hydrochloric acid normal; having examined hectic, recurrent intermittent and typhoid fever patients. (L. Edinger, zur Physiologie u. Pathologie des Magens. *Deutsch. Archiv. f. Klin. Medizin*, Bd. 29 S. 555.) G. Klemperer (Dyspepsie d. Phthisiker *Berlin, Klin. Wochenschr.*, 1889) and Schetty (Untersuchung über Magenfunction bei Phthisis *Deutsch. Archiv f. Klin. Med.*, Bd. 44, S. 516.) confirm the finding of Edinger. Ewald (*l.c.* P. 301) found almost normal digestive power in a case of facial erysipelas. From these studies it is plain that not in all cases of secondary acute gastritis can we attribute the stomach affection to the functional disturbances which the primary disease produces, for in the first place these may be entirely absent, secondly the

frequency of the gastritis is not at all dependent upon the height or intensity of the fever. Thirdly the secondary sympathetic gastritis may set in concomitant with the fever or even before it, ushering in the main infectious symptoms as a prodromal affection.

The secondary sympathetic gastritis is therefore more likely to be originated by localization of the specific, organized disease producers of the fundamental disturbance in the mucosa of the stomach or even by the toxic metabolic products of these microbes. In addition to the infectious diseases already mentioned, this sympathetic form may be a consequence of diseases of the heart, lungs, kidneys and liver, causing venous, passive congestion of the gastric mucosa (Stauungs Katarrh). In cardiac and nephritic diseases the passive gastric congestion may be relieved by appropriate medication directed to the fundamental disorder, *i. e.* the use of digitalis, strychnin and diuretics.



PATHOLOGICAL HISTOLOGY. According to Orth (*Specielle Pathology. Anatomie*, Bd. i. S. 702.) our knowledge concerning the pathological histology of the exsudative inflammations of the stomach is very limited. In the first place, because uncomplicated simple acute gastritis rarely ends in death, and secondly, because post mortem changes and autodigestion exert a most disturbing and disfiguring effect in these superficial diseases particularly. In a case which M. Laboulbène observed, 24 hours after death by rupture of an aneurism, there existed hyperæmia of the mucosa, localized ecchymoses, swelling of the mucus aveoli and augmentation of the mucus.

Delatfield and Prudden give essentially these same changes (*Text book on Pathology*) also Ziegler (*Lehrbueh d. allg. und spec. Pathol Anat.* Yena, 1890.) which may be summarized as follows; The surface of the mucosa is covered by a tough, glassy, cloudy or reddish mucus. The mucosa itself is injected swollen and characterized by a hyperæmia which is limited generally to the py-

loric region and rarely extends to the entire mucosa. Red spots either well circumscribed or diffuse are very evident and ecchymoses are scattered throughout the mucus membrane, larger sugillations occur also but are rare.

The histological changes are by most German authors said to be out of proportion to the degree and intensity of the symptoms (Fleiner *l.c.* P. 233.) That is to say they expect a greater extension and degree of inflammation to correspond to the severity of the symptoms, and are surprised not to find it. Fischl asserts this particularly of the gastroenteritis of children (Fleiner *l.c.* P. 233.) However the exact and very instructive investigations of Prof. Wm. D. Booker (Johns Hopkins Hospital Reports, vol. vi, p. 159—258, Plates xvi to xxi.) show quite the contrary. Booker's researches demonstrate, destruction of the superficial epithelium in parts, infiltration of the mucosa with polynuclear leucocytes, many cover (*oxyntic?*) cells are without nuclei and show only loose, granular protoplasm remaining. Epithelial cells and fragments of glands are collected in heaps on the surface, but not to so marked an extent as in the intestine (Booker *l.c.* P. 251.) In a few cases of acute gastritis associated with enteritis he found the entire gastric mucosa destroyed. Bacteriological cultures were made in 23 cases, in 19 the colonies were very numerous, in 2 moderately numerous and in 2 there were no colonies of bacteria, but many of *ordium albicans*. Tabulated his results appear as follows:

	Predominant.	Numerous.	Few.	Absent.	Pure culture.
	Cases.	Cases.	Cases.	Cases.	Cases.
<i>Oidium albicans</i> .	3	8	1	14	0
<i>Bacillus coli communis</i> .	5	8	4	6	0
<i>Bacilli lactis aerogenes</i> .	7	2	5	7	2
<i>Proteus Vulgaris</i> .	3	0	2	18	0
<i>Streptococci</i> .	0	4	3	16	0

Booker, like A. Czerny and P. Moser concludes that the gastro enteritis of children is a general infectious disease with auto intoxication in which other organs of the body participate, either as a result of an invasion of the body by bacteria, as is often the case with the lungs, or from the effects of poisons absorbed from the gastro intestinal canal. This infantile digestive affection is undoubtedly a more severe and acute disease than any gastritis that occurs in adults, but its study certainly aids our knowledge of the allied

pathological states of adults. There are a number of inflammations occurring in adults as well as children that are followed or preceded by digestive disorders, the etiology of which is much cleared up by the work of the authors above mentioned. I refer to the obscure attacks of parotitis, tonsillitis, pharyngeal abscess, sometimes followed well defined gastric ulcer and the disorders of the heart and nervous system concomitant or succeeding gastro intestinal lesions.

These secondary attacks at times may be autointoxications, then again they show the unmistakeable signs of direct infection secondary to digestive trouble, for in the superficial epithelium is to be found the chief protection of the mucosa against the invasion of bacteria. When the epithelium is well preserved, bacteria are not found in the mucosa beneath, whereas they may be seen entering it where the epithelium has been lost or injured. (Booker *l.c.*) The first step in the pathological process is probably an injury to the epithelium from abnormal or excessive fermentation in the stomach or from toxic products of bacteria and the many other conditions that have already been described. To prevent the effects of auto digestion and post mortem digestion on the gastric mucosa, Ewald suggested washing out the stomach immediately after death and filling it with alcohol. This may in future save a large number of futile investigations. Formerly one depended largely on the studies of gastritis experimentally produced in animals for recognition of the pathological changes. *Thus Ebstein produced gastritis by injecting absolute alcohol into the stomach of dogs.

*Ebstein. Ueber d. Veränderungen d. Magenschleimhaut durch Einverleibung von Alcohol u. Phosphor., Virchow's Archiv, Bd. 55, S. 469.

Losch. Ueber die nach Einererkung abnormer Reize auf die Magenschleimhaut auftretende pathologische anatomischen Veränderungen. Allgemeine Wiener med. Zeitung, 1881, No. 50.

P. Grutzner. Neue Untersuchungen über Bildung und Ausscheidung des Pepsins im Magen. Breslau, 1875.

Edinger. Zur Kenntniss der Drüsenzellen des Magens, besonders beim Menschen, M. Schultzer's Archiv, Bd. 17, S. 209.

C. Kupffer. Epithel und Drüsen des mensch. Magens. München, 1883.

R. Virchow. Der Zustand des Magens die Phosphorvergiftung. Virchow's Archiv, Bd. 31, S. 388; Klebs. Handbuch d. Patholog. Anatomie, 1868, S. 174; Menassein, Chem. Beiträge zur Fieberlehre, Virch. Arch., Bd. 55, S. 452; Ufflemann. Beobachtungen an einem Gastrotomisten. Deutsch. Arch. für Klin. Med., Bd. 26, S. 441.

Marfan. Troubles et lésions gastriques dans la phthisie pulmonaire. Paris, 1889; Stintzig, Münchener med., Wochenschrift, 1890.

A. Sachs. Zur Kenntniss der Magenschleimhaut in Krankhaften Zuständen, Arch. f. experiment. Pathologie, Bd. 22, Heft 3, and Bd. 24, Heft 1-2.

Ewald and Ebstein describe a granular, cloudy swelling in the superficial epithelium. Whilst there is no differentiation possible between the parietal or oxyntic and the central, chief or ferment cells, both varieties are either swollen or contracted, granular, cloudy and with very indistinct nuclei. Between the different epithelia and in the interglandular connective tissue there are considerable masses of round cells. In these as well as in the immigrated leucocytes and the cylindrical superficial cells numerous kariokinetic figures are very evident, and were claimed by Sachs [*l.c.*] to be characteristic for acute gastritis but this is denied by Ewald.

Beaumont gives some strikingly correct descriptions of the conditions observed in the stomach of his patient Alexis St. Martin, when it was acutely inflamed in consequence of overfeeding or of abuse of alcoholic beverages. He states that the mucosa was mostly very hyperæmic even when no digestion was going on, swollen and covered with a thick layer of tough mucus. After ingestion the food was not digested but remained in the stomach from 4–6 hours. The secretion which was much diminished was only rarely weakly acid, mostly it was found alkaline or neutral. After a few days the mucus became still thicker but the hyperæmia grew less. The following account of Beaumont on the state of the mucosa in gastritis: "Its surface was marked with numerous white spots and vesicles like coagulated lymph, between which were very dark red spots:" Is considered by Fleiner [P. 232. *l.c.*] and Fleischer [P. 802, *l.c.*] as unintelligible in the light of our present knowledge. These remarks of the American pioneer of gastric pathology considered in that very light impress me as surprisingly acute and inspire the later days student with respect for the powers of observation in the man. Fisch, [*Fleiner's Lehrbuch*, p. 233] after what he considers very detailed and careful investigations, differentiates three forms of gastritis in children first, an interstitial gastritis which he supposes to start from the connective tissue; secondly, a parenchymatous inflammation having its seat in the glandular tubes; and thirdly, a combined parenchymatous interstitial inflammation. The interstitial affection may be interglandular or submucous also.

SYMPTOMATOLOGY AND COURSE:

Immediately after gross insults to the gastric physiology characteristic signs and symptoms appear. There are fullness in the epigastrium

which is distended and painful to pressure. Eructation, which at first may bring relief later on increases so as to be a great annoyance. Thirst, anorexia and even disgust for food may accompany this. The tongue is often thickly covered with a tenacious white fur, retaining the impressions of the teeth and colored by food or drugs, the breath is offensive. The secretion of saliva is augmented, the pulse small and rapid. There may be painful contractions of the oesophageal musculature, spasmodic yawning and herpes labiales. A burning pain in the epigastrium which may radiate to the hypochondriac regions arises under the sternum (Pyrosis) toward the throat causing burning all the way and sometimes raising sour or bitter stomach contents. As water and other liquids diminish the gastric burning, the patients usually show great thirst, the appetite however is absent or there is a perverse craving for piquante, acid or salty foods, whilst the habitual diet is detested. Taste is much disturbed. The nervous symptoms are general malaise, indisposition to mental or bodily work, prostration, cerebral pressure and a frontal headache. Palpitation of the heart, giddiness, a feeling of fear with profuse sweating are sometimes present. Nervous and less resistant patients (children) may have delirium. Fleiner declares, that general convulsions or loss of consciousness are not rare in his experience.

All these symptoms may arise directly from the stomach or reflexely from the central nervous system, which in these cases suffers intensely at times from the absorption of toxins from the stomach. If the nausea increases to emesis, there will be at first vomiting of food that has been eaten many hours before. This vomited material is mostly badly digested and imbedded in mucus. After emesis the symptoms may ameliorate and the nausea cease, very frequently however the vomiting continues when no more food is in the stomach. Then under much wrenching saliva, mucus, bile and even blood may under great suffering be forced up. Intestinal parasites have in this way been forced into the stomach and vomited up. Skoda first directed attention to cases in which vomiting was much impeded (at times prevented) by spasm of the sphincters, at the cardia particularly.

If the last meals contained an abundance of carbohydrates or fats the vomited material will on testing show an abundance of

lactic, butyric and fatty acids generally; it will also contain acetic acid from the alcohol which was either the cause of all the difficulty or which in 9 cases out of 10 will be administered by laymen:— but the most characteristic chemical condition is the entire absence of free hydrochloric acid in the vomited matter, which is the cause of the perverse fermentations and decomposition in the gastric contents.

The occurrence of sulphuretted hydrogen in the contents of the stomach and in the urine which has been reported by Senator, indicates a condition of albuminoid decomposition which is extremely rare.

STATE OF THE URINE.

The quantity is as a rule diminished, in febrile cases the specific gravity is high and when constipation is present it contains an excess of indican.

FEVER.

Whilst about one half of the cases transpire without rise of temperature in the other half fever is present appearing suddenly and reaching at times 105° F. (40° Ct.) This form may in the beginning occasion some difficulty in the diagnosis because of its strong resemblance to developing enteric, (typhoid) fever. Some German writers still speak of *Gastric fever* as an infectious disease peculiar to itself (see F. Schmidt, dissertation, Berlin, 1885, Z. Frage. d. Existenz. d. Gastrisch; Fiebers, als einer eigenartigen Krankheit.)

Though it is difficult to furnish proof of a direct infection in these febrile forms at present, it is not at all impossible that such a gastritis may exist. Future bacteriological studies in this disease may throw much light on this point. The fever of acute gastritis is usually preceded by repeated chilly sensations or by a typical shaking chill.

DURATION.

If the rules of hygiene are regarded and the patient observes a careful diet, the disturbances will disappear in 3 – 4 days entirely, there are of course much shorter attacks. The stomach remains very sensitive to errors of diet, etc., for a varying time. A number of neglected cases, or those occurring in very weakened individuals, may by a gradual transition turn to the subacute or chronic form.

THE DIAGNOSIS.

In cases that are not accompanied by any fever there should be no difficulty in determining the nature of the disease, especially as the direct cause is in most instances apparent. The febrile form may be confounded with beginning enteric fever, during the first three days of the attack it may be impossible to differentiate the two. The existence of fever blisters (*Herpes labialis*) which according to Leo (*l.c.* P. 66) speaks against typhoid is in my experience an unreliable sign, the results of the blood examinations are contradictory, and in the urine no diagnostic feature is known. The diazo reaction of Ehrlich even when performed in the originators latest method (*Charite Annalen* 1886. B. ii). has in my experiences been of no diagnostic value. In this respect I can confirm the opinions of v. Yaksch and Eichhorst (*Klinische Untersuchungs methoden* P. 777). Most infectious diseases (see above) are in the beginning accompanied by an acute gastritis, in most of them particularly, the exanthemata a differentiation is not difficult. It is a good advice that v. Leube (*Specielle Diagnose*, 1 Theil, Leipzig). gives, when he says: "In all cases with high fever think of other sources and causes before settling upon gastritis". There are two conditions which as far as can be judged at present are reliable factors in the early diagnosis between acute gastritis and enteric fever. (The early diagnosis is the only one I am here discussing, the element of time is very important here as simple gastritis is only of 3 days duration). And these are the manner and rise of the fever and the state of the spleen.

In Enteric fever we mostly meet with a gradual rise of temperature and a gradual fall when the fever subsides. In gastritis the temperature rises abruptly, the remission are slighter and the fall is more sudden. (See Osler, *Principles and Practice of Medicine*, p. 349).

Therefore frequent regular thermometrical studies are not to be omitted. The second diagnostic sign of value is the presence or absence of splenic tumor, its presence points to enteric fever. Unfortunately the splenic enlargement is not invariably present in enteric fever in the outset.

THE PROGNOSIS.

Speaking generally, the prognosis of simple acute gastritis—

except in very old patients and in young children—is favorable.

THE TREATMENT.

1. Prophylactic 2. Dietetic 3. Medicinal.

Prophylactic treatment will especially be applicable to cases that are known to have enfeebled digestive organs or in whom attacks of digestive disease have repeatedly occurred. Attention must be directed to avoidance of injurious influences that may affect the stomach directly from external causes and those that effect it from internal causes.

(a) The external causes are of course the manifold varieties of trauma that are possible in modern life, not only those that can occur accidentally but those that occur gradually by pressure upon the abdomen from without such as is requisite in the execution of certain trades, the manipulation and handling of machines and even the continuous pressure of tables.

A very important matter in this respect is clothing, particularly that of the female sex. Female clothing of today, as far as the maintenance of healthy digestive organs is concerned is not at all conformable to this object. The much condemned corset is not even the worst part of the female outfit for a properly constructed and correctly applied corset need not necessarily effect damage, however for the greater number of ladies wearing them it would be more hygienic to discard it altogether and preserve form and insure support to the breast and graceful carriage in the style of the ancient Greeks, *i.e.* by broad, soft bandages applied immediately to the skin, over the underwear or even externally (*Julia Marlowe Style*.) A more harmful thing than the corset is the tying of the skirts and dresses around the waist.

The most judicious female clothing conformable to the object of relieving the abdominal organs of pressure would be represented by garments made in one piece, of which the upper part supports the lower from the shoulders. (Kleinwächter, *d. Med. Zeit.*, 1894, S. 82, also Meinert, Volkmann's *Klin. Vorträge*, 115, 116.)

The abdomen should always be kept warm not by special bandages, but garments that are made of wool, fitting quite comfortably to the skin and closed below. All digestive sufferers should take special care against cooling or sudden chilling of the surface.

(b) The internal causes of injurious influence must chiefly be

avoided in the food. Exclusive of corrosive and irritant poisons that may be swallowed accidentally the food articles may contain adulterations in form of organic or inorganic additions that are incompatible with sound digestion, or the food may be decayed, fermenting or decomposed. Among the adulterations might be mentioned that of

MILK with water, sodium carbonate and bicarbonate, borax, Salicylic acid, or it may contain bacteria (Tubercle and typhoid bacillus).

CHEESE adulterated with decomposable gelatines and may contain lead and tin from the packing, and also mineral impurities.

SAUSAGES may contain flour, fuchsin, (for coloring) organic poisons, bacteria, ptomaines. (Botulismus poisoning by Sausage.)

BUTTER may be adulterated by mineral substances, gypsum, lime, coloring matters, lead chromate, kresol and binitro naphthol.

VEGETABLE FOOD;
Flour has been found adulterated by sand, gypsum, alum, and also mixed with the fungi of the rye or wheat—Ergot poisoning by rye flour has been observed in Russia, some confectioners use dye stuffs of various kinds all of which are dangerous. Coffee is sometimes adulterated with copper or lead salts to give it a desired color. Wine beer and whiskey are subject to numerous adulterations to effect cheaper manufacture, to preserve or color or give any desired taste. In beer, picric acid, colchicum and strychnin has been found as substitutes for hops, impure grape sugar for malt, alcalies to prevent souring and salicylic acid to preserve it or check fermentation.

Furthermore the prophylaxis must be directed to the (1) quality, (2) quantity of the food, (3) the proper preparation of the food by chewing and insalivation and proper conduct after eating.

These subjects are best studied in works on dietetics:

Gilman—Thompson. *Dietetics*.

Wegele. *Dietetische und Medicamentöse Behandlung der Magen und Darmkrankheiten*.

Penzoldt. *Handbuch der Speciellen Therapie innerer Krankheiten*, vol. iv.

Beidert u. Langermann. *Diatetik u. Kochbuch*, Stuttgart, 1895.

J. Burney Yeo. *Food in Health and Disease*, Phila., 1897.

DIETETIC TREATMENT:

Acute inflammation of any structure is best treated by rest and the stomach forms no exception (Tompson). Hence total abstinence from food and great reduction of the quantity of fluid imbibed is often curative after an interval of thirty-six hours. So, for the first two days as little food as possible should be allowed. To accomplish this very simple and logical object is in private practice a most difficult thing. There is an incorrigible custom, among relatives to stuff the patient with all manner of articles, which is hard to combat. At the bottom of all of this probably lies the popular superstition that a human being can not exist twelve hours without food. A total abstinence from food is born very well and leads most rapidly to recovery. For the intolerable thirst, cracked ice should be given, a wineglass full in 2 hours. If there are signs of collapse, champagne or brandy can be added with safety even if alcohol was the cause of the trouble.

After the 24 hours of total abstinence, the first food to be given is milk, beef bouillon with soft rice or an egg beaten up in it. A good stimulating food when there are signs of prostration consists of one raw egg beaten up with half a pint of Hocheimer or a full pint if desired and sweetened to taste with a slight flavor of lemon added. The wine may have to be diluted if the gastric mucosa is very sensitive. Of the above a wineglassfull may be given every 2 hours, (quite warm if preferred). On the third day a few soda crackers, or cakes may be allowed. On the fourth day a gradual return to more reconstructive food is advisable such as calfs brain free from all stringy and membranous parts, boiled first in bouillon then rapidly broiled thereafter, sweet bread or thymusgland broiled, breast meat of broiled squab, pigeon or chicken. Finally, on the sixth day after the attack, finely scraped broiled beef, potato purée, stewed apples, rice, tapioca, very soft omelette.

MEDICINAL TREATMENT:

Acute gastritis must be treated without drugs wherever it is at all possible. If the dietetic rules of total abstinence from all food for 24 hours and cautious return to light diet are carried out, two thirds of the cases will recover without medicines. Not a few patients, even children will do this instinctively and not permit any cramming with food until the stomach has become rested and a

natural desire therefore returns. The most important indication of treatment is usually done by the injured organ itself, *i.e.* evacuation.

If emesis does not occur easily at the outset, both Ewald and Boas recommend the following emetic;

R		
Pulvis Ipecacuanhæ,	1.5	Gr. xxiii.
Antimonii et Potassi. tartratis	0.05	Gr. five-sixths.
Sig.		Misce.

Fiat Chart, No. 1,—to be taken at once or in divided doses.

In children, Ewald favors a teaspoonful of the syrup of Ipecac. I have so far been able to accomplish all that was necessary without emetics and loath to advise their use. Where emesis must be brought about it is more expedient and reliable to use $\frac{1}{4}$ Gr. apomorphine hypodermically. Another drug which gives satisfaction to both patient and physician in this attack, particularly when there is constipation, is Calomel. Sometimes when persistent nausea follows thorough emesis it may even act as a gastric sedative. Ewald advises Gr. vi repeated in an hour. Whilst this dose seems large, it is by no means too large and will produce a cholagogue and infecting effect that may terminate the gastritis then and there. Formerly I used tablet triturates of Gr. $\frac{1}{2}$ of Calomel every hour until purgation, they are more pleasant to administer. The larger dose recommended by Ewald produces more of an antiseptic action since a portion of it is converted into the mercuric bichloride.

Calomel can not be given at the beginning of the gastritis very well, the second day is best suited for its administration. Although I mention these drugs it is not with a view to routine treatment but to aid in meeting special indications. When pain in the stomach is attended by chilliness, hot poultices over the entire abdomen, turpentine stupes or spongiopiline dipped into hot water and 10-20 drops of Tr. of opium sprinkled over before it is applied to the epigastrium. But when there is gastric pressure that seems to embarrass respiration and associated with explosive eructation, cold hydropathic applications are more effective. When there is fever these applications should be made with ice water or the ice bag. Intense pain is met with hypodermic injections of morphine Gr. $\frac{1}{4}$ and atropiæ sulphate Gr. 1-150. The following suppositories of Boas can

R Codein phosphoric 0.05
Ext Bella dounae 0.03

find application f. c Butyr. Cacao suppositor. N^ox. One every hour until relieved. Where the pain must be relieved and the hypodermic injection is not permitted and medication per os not retained.

By the mouth Codeine is best given in the following manner;

R Codeine phosph. 0.1
Sig. Aquae Menth pip. 40.0

One teaspoonfull every 3 hours. M.

If symptoms of hyperacidity, keeping up the annoying pyrosis and thirst are predominant it may be impossible to avoid alcalies. They are expediently prescribed in the succeeding form.

R Magnesia Calcined
Sodium bicarbonaie a.a. 10.
Menthol 0.2.

½ teaspoonful pro re nata. Mix thoroughly.

It is not rational to give purgatives because they irritate the inflamed mucosa, Calomel is the only drug of this nature that can safely be given, but not before the fermenting stomach contents have been removed by emesis or lavage. To effect purgation before the stomach is emptied, exposes the intestine to infection from the septic mass forced through it. Persistent vomiting may call for especial treatment here morphine hypodermically, mustard plasters to the epigastrium and small pieces of ice will be sufficient. A singular case of very exhausting and persistent vomiting was in my practice relieved by Cocaine hydrochlorate Gr. ½ Menthol Gr. Aquae Camphor fl ʒss. M. every 2 hours until relieved. Vomiting of this character is bound to bring on collapse, it is fortunately a rare complication but must be met energetically if it develops. In concluding the Medicinal treatment I desire to refer to a succesful therapeutic measure which does not properly belong under this heading, because it is not medicinal, but mechanical.

This consists of evacuating the stomach with the tube and immediately thereupon disinfecting it by washing it out with a solution of the following composition.

R Thymol Gr. viii.
Acid Boracic ʒii.
Warm water Oi (one pint).

The water during lavage must be used quite warm and the antiseptic not used until the plain water runs out clear. Catch up the outflowing antiseptic fluid and ascertain that it approximates one pint, a few ounces retained will not do harm. Vomiting as a rule ceases entirely after this. Six hours later wash out the color by large enemata of 20 % solution of Boracic Acid, no matter whether the patient has diarrhoea or constipation. If diarrhoea exists it is absolutely rational to effect the removal of the putrefying colonic contents by large enemata, (given in the knee chest position) and if constipation exists the stagnation of feces certainly aggravates the symptoms by increasing flatulence and abdominal pressure. If there is any therapeutic measure in addition to abstinence from food that merits confidence it is this mechanical cleansing of stomach and colon. Rare cases of high temperature may need special therapeutic measures for the fever; Here also drugs must be avoided and the temperature reduced by sponging with cold water or the cold bath.

In case the appetite fails after the attack or there is protracted weakness with timidity and aversion to food, the following tonic may become necessary.

R	Strychnin Sulphatis	Gr. $\frac{1}{3}$.	
	Acidi hydrochlorici dilut.	fl. $\overline{3}$ ii.	
	Elixir Gentianæ qis ad Mis.	fl. $\overline{3}$ vi.	
Sig.			Misce.
	One tablespoonful $\frac{3}{4}$ hous before meals through a glass tube.		

PHLEGMONOUS OR PURULENT GASTRITIS — SUPPURA-
TIVE INFLAMMATION OF THE STOMACH
— GASTRIC ABSCESS.

THIS is a very acute, fatal, and fortunately, very rare affection of the gastric walls apparently set up by an invasion of pyogenic cocci. It is a purulent inflammation invariably originating in the submucous connective tissue and from here extending to the mucosa. Ziegler (*Lehrbuch d. Allgem. u. spec. Path. Anat.*, 1887. Bd. ii. S. 516) describes large numbers of streptococci occurring partly free in the tissues and partly in the protoplasm of the cells. In case the serosa is invaded the disease as a general thing produces a general fatal peritonitis by perforation, unless an infection of the peritoneum is prevented by an agglutination with adjacent organs. I have never seen a case of this sort but as far as one is able to judge from the literature of this subject the disease is inevitably fatal running most always an acute, rarely a subacute course. Ewald (*l.c.* P. 303) has seen only one case and that at the clinic of his teacher Frerics. It occurs much oftener in men than in women; of 41 cases 33 were men and 8 women. In a report by Glax, (*Die Magen-entzündung Deutsch. Med. Zeit.* 1884, No.3) it is stated that but 51 cases had been observed up to that time. Most authors that have had experience with the disease, distinguish first, an idiopathic primary purulent gastritis, the etiology of which is obscure and second-

ly, a secondary, metastatic phlegmonous or purulent gastritis which is an accompaniment or a sequence of other infections such as pyaemia, puerperal fever, anthrax, typhus or Variola. Anatomically one may distinguish a diffuse and a circumscribed purulent inflammation of the submucosa, the latter is spoken of as stomach abscess.

ETIOLOGY;

The direct cause of the rarer idiopathic phlegmonous gastritis is unknown. The predisposing causes may be the same as stated under the etiology of simple gastritis. The direct causes judging from anatomical specimens are undoubtedly bacterial invasions of the submucosa probably, principally, by pyogenic cocci, that find portals of entry through lesions in the superficial epithelium of the stomach, such as occur in most all gastric diseases, especially in so-called exfoliation in old ulcers or after trauma caused by fish bones, seeds, foreign bodies, etc., Ziegler's (*l.c.*) studies have already been mentioned. The secondary, metastatic phlegmonous gastritis which seems most frequent is that following puerperal fever and owes its origin to localization in the stomach of the specific organisms producing the fundamental disease. Whatever they may be it is selfevident that only an enfeebled organ is liable to such an inflammation.

PATHOLOGICAL ANATOMY:

The diffuse inflammation rarely invades all parts of the stomach with the same intensity, even if the whole organ is involved. The pyloric portion is generally invaded more than others; toward the cardia the inflammatory process is less and less marked whilst, the oesophagus is rarely attacked. The submucous layer is most extensively altered on cross-section, it is swollen showing an oedematous, purulent, or at times, a bloody infiltration. From here the inflammation spreads along the interglandular tissue between the glandular tubules, effecting fine or larger perforations in the mucosa which may assume a sieve-like appearance. Pus wells up through these cribriform perforations as out of a swollen sponge. It may occur that the mucosa is lifted from the submucosa by accumulations of pus. Rokitsansky has described a case in which the mucosa was only strikingly anæmic, otherwise unaltered. Macleod (*Lancet*, 1887, vol. ii, P. 1166) describes a gastric abscess in which mucosa was said to be unaltered.

Toward the deeper portions of the engorged layers, the process spreads along the bundles of muscular fibers in the muscularis, which undergo fatty degeneration and show infiltration with pus cells and proliferation of nuclei. The serous or peritoneal layer may also be lifted from the subserous or muscular layers and perforation as a rule rapidly follows inflammation of this layer. Circumscribed abscesses which must be differentiated from the diffuse inflammation are usually small, varying from the size of a hazelnut, to that of a goose egg (Leube *l.c.*). On cutting into the swollen elevated areas of mucous membrane, the abscess is found in the submucosa but may extend through the muscularis to the serosa.

SYMPTOMATOLOGY;

The symptoms are very much like those of a very intense acute simple gastritis; the pain of gastric phlegmon is not materially increased by change of position or pressure. There is very rarely any vomiting of pus in diffuse purulent gastritis. Gastric abscess may be attended by copious vomiting of pus, after which a tumor that may have been palpable before, may become much smaller, or disappear entirely; this phenomenon might be significant for the diagnosis of gastric abscess if it were not for the fact that pus tumors of the neighboring organs, sometimes break through into the stomach and cause the same symptoms. The fever reaches 104°–105° F., the patient being aware from the outset that he is very seriously ill. The sensorium is much disturbed by great restlessness, headache, insomnia, delirium. To the symptoms of acute gastritis those of a sudden peritonitis may be added later on.

DIAGNOSIS:

The important conditions for diagnosis are the pain, vomiting, meteorism, fever, diarrhoeas and general phenomena. The pain is localized in the epigastrium and said to have been absent in some cases. The emesis is always present and consists of bile, mucus and food *debris*, in diffuse purulent gastritis, pus has not been noticed in the vomit, which strongly resembles so-called peritoneal vomiting.

The fever is very high and the temperature curve is said to resemble those of pyæmic fevers with marked remissions and exacerbations. Tympanitis and diarrhoea are more frequent than constipation. Other symptoms are, rapid compressible pulse, cold peripheral parts, hurried respiration, thirst and a much coated tongue. The

course of gastric abscess is not characteristic and Leube states (*Spec. Diagnost. Inneren Krankheiten* S. 237) that the diagnosis is a matter of chance. The attack may resemble a circumscribed peritonitis or one of the various perigastric inflammations or abscesses, according to Ewald (*l.c.*) it may so mimic abscess of the spleen or left lobe of the liver, that a differential diagnosis is absolutely impossible. Deininger (*Deutsch. Archiv f. Klin. Med.* Bd. 23, S. 268.) held that high fever, constant and intense gastralgie pain that is not increased on movement, and increased resistance in the epigastrium, should be sufficiently characteristic to justify a diagnosis. These symptoms however occur also in above conditions referred to by Ewald. Chvostek (*Wiener Klinik*, 1881, and *Wiener med. Presse*, 1877, Nos. 22–29.] however seems to have made the diagnosis in one of his cases. Where there is probability of diffuse or circumscribed phlegmonous gastritis, the exploratory puncture with an aspirating needle or the exploratory incision is in my opinion justifiable. In Penzoldt u. Stintzing's new *Speciel Therapie Innerer Krankheiten*, vol. iv. p. 446, von Heinecke gives suggestions for the operative treatment of phlegmonous gastritis.

PROGNOSIS is almost always unfavorable, especially in the diffuse form. After the circumscribed form, and evacuation of the abscess, several clinicians have reported recoveries, (Deininger *l.c.*, Glax *l.c.*, Kirchmann *l.c.*, also Buckler, *Idio. Path. phlegmon. Gastritis*, Bayer, *ärztliches Intelligenzblatt*, 1880, No. 37.) but it is impossible to confirm whether they were really gastric abscesses. Dittrich has found cicatrices in the submucosa pointing to the possibility of healing.

TREATMENT:

If a diagnosis could be made it seems to me that these cases, the diffuse as well as the circumscribed forms, had best be treated surgically. Under the existing difficulty the treatment must be only symptomatic and limited to relieving pain by hypodermic injections of morphine, applications of ice, ice bag to the stomach, crushed ice by the mouth. To counteract collapse, wine enemata and hypodermic injections of strychnin are recommended. Medicines by the mouth are worse than useless.

INFECTIOUS GASTRITIS — (Gastritis infectiosa, diphtheritica, crouposa, mycotica parasitaria). As Penzoldt correctly remarks.

(*l.c.*) every gastritis is to a certain extent infectious, it is therefore that a number of authors reject the conception of infectious gastritis as a separate and distinct disease. Lebert (*l.c.*) and Osler (article on gastric diseases in *Eulenburg's Realencyclopædie* 2nd edition, vol. 12, p. 410) believe that this is a characteristic infectious gastritis peculiar to itself. Boas (*Speciel. Therapie. d. Magenkrh.* p. 6) is of the opinion that there is a form of acute gastro enteritis, well characterized clinically, which differs from simple gastritis by the gravity of the symptoms and particularly the course of the fever, so that it merits separate consideration. Ewald on the other hand holds that there is no sufficient specificity of inflammatory processes affecting the stomach for establishing a separate class of infectious gastritis. Fleiner, Penzoldt and Einhorn give no separate consideration to infectious gastritis. Those that establish a separate category for this affection class under this head all gastric invasions by infectious germs, so that all forms as remarked before are to a certain extent infectious.

The symptoms are said to be very similar to acute simple gastritis and therefore require no further description. The course is more, protracted as it may last according to Boas 3–10 days, according to Lebert (*l.c.*), some case may have fever for 2–3 weeks. Gaffky (*Deutsche med. Wochschr.* 1892, No. 14) gives an account of severe gastro enteritis in three persons who drank the unboiled milk of a cow affected with hemorrhagic enteritis. Gaffky believes that the infecting germ was a particular virulent type of the *Bacillus coli communis*. A number of similar mass epidemics are on record (Husemann, *Deutsch med. Wochschr.* 1889, S. 960) that tend to strengthen the conception of a special infectious gastritis. There seems no necessity as yet for a separate classification of this kind the subject is still too hypothetical to be ranked as equal in importance with other well characterized forms of gastritis. The diagnosis, prognosis and treatment is said by Boas to be the same as for acute simple gastritis.

Diphtheritic gastritis is a rare affection, occurring not only as a sequence to Laryngeal and pharyngeal diphtheria but also as an accompaniment to pyæmia, septicaemia, puerperal fever, scarlatina, variola, endocarditis ulcerosa thyhus etc. The disease is as a rule not discovered until the autopsy is made and for that reason has

more of a pathological than clinical interest.

MYCOTIC GASTRITIS:

When the vitality of the mucosa and the secretion of hydrochloric acid has been reduced, suppressed or destroyed certain pathogenic fungi are known to invade the mucosa, producing ulcerations and necrosis.

Most of these mycotic gastritic inflammations can not be recognized during life as such. Those that have been described are the anthrax gastritis produced by spores or bacilli of anthrax lodging in the mucosa or submucosa and giving rise to inflammation, ulceration and necrosis.

Sidney Martin observed a case of anthrax of the anterior wall of the stomach at Guy's hospital, the primary infection was in the left cheek where a malignant pustule developed (*Journal of Pathology and Bacteriology* vol. i).

Gastritis caused by the favus (fungus achorion Schönleinii) has been reported by Kundrat (über gastro enteritis favosa *Wien. med. Blätter*, 1884 No. 49). The case was that of a drunkard whose gastric mucosa was predisposed by alcoholic chronic catarrh, he had favus universalis and in the stomach and intestines the fungi had caused diphtheritic inflammations, with fibrinous exudations, ulceration and sloughing, death was caused by a terminal colitis it appears.

The thrush fungus German-Soor Latin-*oidium albicans* has been reported as setting up a mycotic gastritis; in some cases of which the stomach alone appeared infected, throat and oesophagus were intact.

The yeast fungus (*Torulae* or *saccharomyces cerevisiae*) *sarcinae*, the common moulds (*Penicillium Glaucum* and *Mucos*) and various *schizomycetes* occur in the gastric contents and set up irritation of the mucosa not by direct invasion it appears but by the toxic products of the fermentation which they cause.

Sarcinae according to Hühne do not bring about any fermentation.

Miller's interesting investigations concerning the bacterial flora of the mouth have been referred to on page 41. In the first volume of his excellent text book (*Specielle Pathol. Anatomie* B. 1, p. 704). Johannes Orth describes an interesting bacteriae invasion near an

old chronic gastric ulcer which had largely become healed. At several places there were grayish, bran like incrustations partly adherent which anatomically had to be designated as diphtheritic. In the scabs or crust and in the deeper parts of the mucosa, and partly, lodged distinctly in lymph vessels, were numerous bacilli that had some morphological resemblance to those of anthrax, this supposition could however not be confirmed by cultures. The case was complicated by the fact that a fatal hemorrhage had occurred from a very small arteriole at a place where only a very tiny defect in the mucosa was observable. In the immediate neighborhood of this defect the bacilli were found also but not insufficient numbers to attribute to their destructive agency the tearing of the arteriole which was not aneurismatic.

Orth then proceeds to refer to the bacillus gastricus or polysporus brevis of Klebs (über infectiöse magenaffectiōnen *Allgemein. Wien. med. Zeit.* 1881, No. 29 and 30) which was found free in the lumen of the glands as well as between the cells of the glands and the tunica propria; there was also an interglandular small round cell infiltration.

Böttcher (*Dorpat. med. Zeitschr.* 1875, p. 184) also defended the view that gastric ulcers are in part due to mycotic and bacterial invasions. Unfortunately Klebs' and Böttcher's statements have not been confirmed by later investigators.

Animal parasites are also on record for producing gastritis; C. Gerhardt (Magenkatarrh durch lebende dipterenlarven *Jenae med. Zeitschr.* Bd. 3, S. 522) gave an account of acute gastritis set up by larvae (maggots) of diptera, a class of insects of which the common fly, the flea, etc., are examples. The egg of these larvae were said to have been swallowed with raspberries. Meschede (Ein fall von Erkrank. durch im Magen weilende lebende Maden *Virchow's Archiv*, B. 36. S. 300) reports gastritis caused by maggots eaten with cheese. Senator reported gastritis set up by living maggots of the common fly which occurred in the mouth and stomach (*Berlin Klin. Wochenschr.* 1890 No. 7) the same observation was made by Hildebrandt (*Berlin Klin. Wochenschr.* 1890, No. 19). Fermaud observed a case of gastritis and gastralgia caused by an earthworm (*Journal de med. pratique de Paris*, 1836, tome vii. p. 57). It is known also that ascarides and tape worms may reach the stomach

in rare cases and give rise to severe inflammations, which may subside at once as soon as the offending parasite is vomited.

TOXIC GASTRITIS: (gastritis venenata)

This form of acute gastric inflammation is caused by poisons or corrosive chemical bodies. The poisons that have been taken either by mistake or suicidal intentions are mercuric bichloride or corrosive sublimate, phosphorus, arsenic chloroform, creosote, potassium chlorate, oxalic acid, nitrobenzol, carbolic acid, the concentrated inorganic acids, sulphuric, hydrochloric and nitric acid. The caustic alkaline hydroxides in strong solution, and furthermore alcohol in all its forms and some substances used as medicines and (see etiology of acute gastritis) particularly croton oil and antimonium and potassium tartrate (tartar emetic) also ammonia.

THE PATHOLOGY will necessarily vary considerably as it is not only dependant upon the kind but also upon the quantity and concentration of the poison, or upon the circumstance whether the poison is taken on a full or empty stomach as food and drink dilute the drugs. There may be only a very slight superficial inflammation or a very penetrating corrosive effect, involving the entire gastric wall and even leading to perforation. Different drugs produce different effects upon the mucosa. Phosphorus, arsenic antimony and alcohol produce in excessively large, toxic doses a milky, yellowish white or opaque appearance. The epithelia of the alveoli of the tubular glands are partly in a state of mucoid degeneration, partly finely granulated, cloudy and showing fatty degeneration; the same is the case with the secreting cells. The tissue between the cells is crowded with a small round cell infiltration. In this condition autodigestion by the gastric juice may cause peptic ulcers *i.e.* when the poisons are not taken sufficiently strong to effect ulceration or to destroy secretion.

Dilute acids and alkalies induce the pathological picture of a simple acute gastritis, whilst in concentrated form, a deeply penetrating necrosis with formation of crusts and intense reactive inflammation with serous infiltration, suppuration and blood extravasation are the result. The scabs or crusts show different colors with different corrosives. Under the effect of sulphuric acid they are black, of nitric acid yellow, of alkalies brown, of copper salts

green or blue, of silver salts black. Dislodgement of these crusts leads to fatal bleeding, tearing off the serosa or perforation with peritonites. Oxalic acid is said to produce a jelly like swelling that is semitransparent, ammonia causes a pustular one.

SYMPTOMS:

After taking the poison there is generally indescribably severe pain, intolerable burning, vomiting which increases the pain and causes fainting at times. The vomit as a rule contains blood or bloody mucus, the thirst is great. There is most frequently diarrhoea containing blood. Severe general symptoms follow, small very fast pulse, jactitation delirium. In case much of the poison has reached the general circulation, haematogenous icterus, petechiae albuminuria and haematuria may follow. Death follows in a few hours or few days from collapse or later by perforation peritonitis. Even if the patients are tided over the first period of acute gastric symptoms, they may die later in from hemorrhage when the scabs and crusts are sequestered or by the consequences *i.e.* stenosis of the oesophagus, cardia, pylorus or atrophy of the mucosa.

THE DIAGNOSIS after knowing the history of the case will not be difficult. One should not fail to make a thorough examination of the mouth and throat where the corrosive effect, if any, will be evident. An analysis of the vomit will probably inform of the nature of the poison.

THE PROGNOSIS of severe toxic gastritis is necessarily fatal if not by the direct poisoning or first destructive effect of the drug, certainly by the severe secondary effects.

THE TREATMENT will vary with the nature of the poison. In recent poisoning with strong acids magnesia usta, (calcimed) should be given as soon as possible, if no drugstore is near, give chalk or even powdered lime which can be scraped from the wall. Whenever possible the stomach tube should be at once used for all poisons, of recent date.

Boas, Fleischer and Pick advise that it should not be used in severe acid or caustic alkali poisoning because of the danger of perforating the stomach. As most such cases will probably die of perforation anyway, I certainly should use the tube and let the patient take his chances. A large quantity of water with sod bicarb in case acids were taken or vinegar in case alkalies were taken, will

doubtlessly dilute and combine with the destructive agent present. Lemon juice will also answer for the alkaline caustics. In all other poisonings the stomach tube, or if convenient the pump should be used as soon as possible and the stomach washed out thoroughly. For other poisons the approved antidotes should be given (freshly prepared hydrated oxide of iron for arsenic, etc.) that will be found in various text books on toxicology and therapeutics; H. A. Hare's system; H. C. Wood; Lauder Brunton; Binz, Schmiedeberg; Penzoldt and Stintzing's system vol. vi. After carbolic acid ingestion, wash out, and then pour in olive oil 250 c.c. In all corrosive poisoning cases the pouring in of olive oil or molten vaseline, after neutralization and washing out, will if possible, diminish the corrosive effect. Where not too much acid or alcaly has been taken, the subnitrate of Bismuth or subgallate of Bismuth, \mathfrak{z} i t.i.d. if possible, swallowed with oil will favor rapid cicatrization and inhibit bacterial infection of the necrosed, charred areas. A suspension of Bismuth subnit. \mathfrak{z} i. to one pint mucilage and water has proved advantageous in a case of carbolic acid poisoning in my practice.

If the pain is severe, morphine hypodermically in $\frac{1}{4}$ to $\frac{1}{2}$ grain doses repeated until relief comes. It is our duty to give relief to the pain at any risk, even if chloroform anæsthesia is required; for after the suffering ceases our efforts to save the patient can be more easily executed. Nutrition must be carried on by rectal enemata only. By the mouth, ice is about all that is permissible; it will tend to diminish the pain, fever and inflammation. I make such an explicit statement of treatment because I had experience with two cases where the autopsy showed that recovery might have been possible as not much sulphuric acid had reached the stomach if the treatment had been more heroic *i. e.* if the tube had been used for timely removal of the poison.

CHRONIC GASTRITIS.

NOT much over a decade ago it was customary to class all stomach diseases that were not acute and that could not be diagnosed as dilatation, ulcer, or carcinoma, under the head of *chronic gastric catarrh*. With Ewald and Penzoldt, I agree in their objections to the word *catarrh* and the reasons have been given under the chapter on simple acute gastritis.

Even at the present day there is no absolute uniformity on the conception and limitations of the word chronic gastritis. With the aid of improved methods of diagnosis, particularly such methods as permit of an exact study of the various gastric functions, the so-called gastric neuroses have been recognized as separate and distinct diseases, formerly they were believed to be symptoms of chronic gastritis. This chronic inflammation of the mucosa affects all of the important functions, although one or the other of these is generally most seriously affected. There are observed many variations of the kind and intensity of disturbed function, from a trivial reduction of secretion of gastric juice or interference with motility to complete suppression of glandular activity and pronounced insufficiency of the peristalsis. There are two pathological processes that appear as conditions to every chronic gastritis; these are desquamation and

degeneration of the glandular cells and infiltration of the connective tissue.

Bearing in mind these conditions we may distinguish two main types of chronic gastritis, first the hypertrophic, and secondly, the atrophic.

The hypertrophic form consists of proliferation of the connective tissue leading to change of form and folding or warty elevations of the mucosa (Etat mammelonné or polyposis). The end of this process is a complete destruction or cystic degeneration of the glands. A grayish brown or in places a dark red brown color is peculiar to this swollen and proliferated mucosa, which is covered with an adherent gray coating of mucus.

The atrophic form consists of contraction of the connective tissue, loss of the epithelium and more or less complete destruction of the glands, in rare instances, superficial ulcerations. The mucous membrane is much thinned out, very smooth and of a grayish white or pale slate gray color. If the process attacks the muscularis and submucosa it may cause atrophy of the muscle fibrils, with or without thickening of the entire gastric wall due to connective tissue new formation. Then again we may meet with a genuine hypertrophy of the muscularis, particularly at the pyloric portion or in the pylorus itself.

The lumen of the stomach in these forms may show a normal capacity. Or it may be much diminished in size by connective tissue thickening of the gastric walls and subsequent contraction, this is known as *gastric cirrhosis* (Brinton) and may reduce the normal capacity to one of 160 c.c. (Leube, Penzoldt). Or again, the capacity is much increased by a dilatation in consequence of chronic gastritis and hypertrophic pylorus stenosis.

So the anatomical picture may present, atrophy of the mucosa with wasting of the tubular glands and of the muscularis, thinning of the entire gastric wall and very frequently dilatation, or on the other hand, inflammatory hyperplasia of the layers of the stomach with excessive connective tissue proliferation leading to cirrhosis ventricul hypertrophic pylorus stenosis, atrophy of the glandular layer and sometimes of the muscularis.

This form may lead to marked reduction of the lumen or more frequently if a stenosis exists, to a dilatation. Both forms

bring about grave disturbances of motility, secretion and absorption. The cause of the elevated, warty or polypoid projections of the glandular layer is to be sought in the fact that in certain forms of the disease the mucus layer grows much more rapidly than the submucus layer, bringing about a rough, wrinkled, mammillated surface that has been described as gastritis polyposa, and by some French writers is termed *etat mammeloné*. (See Orth, *Specielle Pathol. Anat.* bd. i, p. 709). A number of Germans describe a variety of special forms of chronic gastritis under the names of *Saurer Katarrh*—sour or acid gastritis, *Schleimkatarrh*—slimy or mucus gastritis, also termed *gastritis atrophicans* and a simple chronic gastritis or *Einfacher Katarrh*. All of these terms are quite unfortunately invented and unscientific because they are artificial. The so-called *Saurer Katarrh* is not a gastritis at all, (Ewald) but a neurosis of secretion, a hyperacidity and the result of secondary irritation of the mucosa.

ETIOLOGY

Chronic gastritis is a widely spread disease occurring in all stations of life but more frequently among the poorer classes where the quality of the food is so inferior as to keep the stomach in a constant state of irritation. All the numerous injurious influences which arise from a defective and inappropriate diet have been referred to under the head of the pathogenesis of acute gastritis. It may evolve out of the acute or subacute form as the mucosa has been damaged by the altered circulation and its resistance to disease lessened. All processes that lead to venous congestion of the stomach, *i. e.* affections of the portal system, especially of the liver and spleen, also diseases of the heart may cause it. There are certain conditions which may bring about chronic gastritis by effecting alterations in the composition and structure of the blood, these are anaemia, chlorosis, scrofula, secondary anaemias following typhus and typhoid fevers, the exanthemata, pregnancy, tuberculosis, diabetes, gout and Nephritis. Irritating substances brought in contact with the mucosa continuously either from without or within *i. e.* from the blood are believed to cause the disease. Ewald states that it may result from direct local irritation and neoplasms. My experience is that in the vicinity of such structural changes pre-existing in the mucosa, there is indeed a gastritis observable but it mostly partakes

of an acute or subacute type. The defective chewing and insalivation, hurried eating and swallowing of large pieces of food, putrefaction of the mouth from carious teeth or the manifold forms of stomatitis and gingivitis. In this country the excessively hasty eating and the abuse of ice water with meals and of tobacco and alcoholic liquors between meals are the causes of the frequency of chronic gastritis.

The American people residing in cities live for a large part under commercial and social customs that are pernicious to the digestive organs. Foremost, among these conditions are the high mental pressure under which the demands of business are carried on, the constant worry, nervous tension caused by force of competition.

The anxiety to get rich rapidly by straining all mental and physical powers to make the utmost possible gain, all these things bring about a hasty, nervous manner of taking food, chewing is a process which most business men execute in a perfunctory manner only. There is no time for insalivation, if they could they would gulp the food down dry, as it will not go down that way they help it down with ice water. Tobacco juice is responsible for much of this disease, also condiments used habitually, pepper, ginger, mustard, horse raddish and the habitual use of drugs (arsenic, silver salts.).

Chronic gastritis is the most frequent disease among habitual consumers of alcoholic liquors. From what was said under acute gastritis of the experimental production of this disease with alcohol by Ebstein, the frequency of the chronic form among the devotees to Bacchus and Gambrinus is very intelligible. As Ewald correctly remarks the disease may be classified among those in which the patient's indiscretions play a very important role. But as most persons treat their stomach badly and neither eat with proper mastication and insalivation, nor are able to resist culinary temptations, gastritis is one of the *best nourished* and most prevalent diseases in the world. "Indigestion is the remorse of a guilty stomach" says Ewald and F. Albin Hoffmann (*Vorlesungen ueber allgemeine Therapie* Leipzig, 1885.) expresses a sentiment that deserves to be an apothegm — "Jeder Mensch hat den Magen den er zu haben verdient," — "Every one has the stomach which he deserves." It is not intended

to do injustice to a large number of sufferers from weak stomachs who take the greatest possible care to avoid dyspepsia and nevertheless are liable to acute or chronic gastritis. The etiology explains why the male sex is affected much more frequently than the female.

THE PATHOLOGICAL ANATOMY:

These changes are as in the acute form, most pronounced in the pyloric region and from here extends to the fundus. The alterations of structure occurring in the course of chronic gastritis present varying pictures according to the duration of the disease. In the later stages the variations are considerable, since at this period the consecutive changes may one time incline to inflammatory hyperplasia, at another may show an atrophic character and also because either the mucosa or submucosa only or in other instances the deeper layers may be involved with alternating intensity and extent.

The inflammatory process is not at all limited to mucoid degeneration and desquamation of the surface epithelium but pre-eminently affects the glandular elements and interstitial tissues and from here attacks the deeper layers of the gastric wall. In early stages there is a general diffuse redness, in later stages this color that is due to hyperaemia gives way to a peculiar pigmentation which first assumes a bluish or brownish shade and finally gets to be of a dirty red brown or slate gray or both. This pigmentation is generally limited to the pyloric region but in spots it may be spread over other sections of the inner surface of the stomach. The color is due to blood pigment which has become stored up in the cell and interstitial tissue; also to blood corpuscles that have left the vascular channels and undergone pigment metamorphosis during the long standing chronic hyperaemia. This pigment action must not be confounded with post mortem discoloration.

INFLAMMATORY HYPERPLASIA:

In this form the gastric mucosa may either preserve the velvety appearance that is peculiar to the normal inner surface of the contracted stomach or it may be covered with irregular warty projections and exhibit immense development of the pyloric *plicae villosae*; this is due to inflammatory infiltration of the interglandular and subglandular connective tissue, but particularly to the same process occur-

ing in the connective tissue ridges present between the vestibular entrances to the gland ducts (*Vorraume of the Germans*), or peptic duct alveoli as I prefer to call them.

If these hypertrophic — hyperplastic processes are confined to circumscribed spots, they may assume exaggerated degrees forming polypoid proliferations, which as a rule are attached by broad bases, but in consequence of connective tissue contraction they may occur pedunculated. In this way papillomatous excrescences may be developed which project into the lumen of the stomach (gastritis polyposa, Orth, *l.c.* p. 716). When the submucosa is attacked with inflammatory infiltration and connective tissue new formation, the loose tissue is first transformed into one much richer in cells and then into a tougher, more inelastic layer resulting naturally in a much reduced movability of the mucosa upon its substratum. When this chronic process leads to cicatricial contraction in the hyperplastic submucous tissue it may lead either to partial, localized change of form or to a more or less general, uniform contraction (*Schrumpfung, cirrhosis ventriculis. Linitis plastica, Brinton*). In the pyloric portion this process may lead to stenosis. Frequently the muscularis is hypertrophied as a consequence of the chronic irritation transmitted through the submucosa, this muscular hypertrophy is most pronounced at the pylorus.

This localization of the maximal intensity of the inflammatory process in the mucosa, submucosa and the muscularis at the pylorus makes the origin of a pylorus stenosis in consequence of chronic gastritis intelligible. This kind of a stenosis is usually spoken of as benign, in contradistinction to the malignant stenosis of carcinoma.

Much diversity of opinions exists concerning the origin of the *état mammeloné* (*Mammelon* means the nipple of the mammary gland. *Frerichs* held that it was due to accumulations of fat in the mucosa and inflammatory hyperplasia of the lymph follicles contained in it. *Rindfleisch's* view was a greater increase in growth of the mucosa than in the submucosa. *Ziegler* explained the mucosa polyps by proliferation of the submucosa. *Ebstein* assumes an inflammatory hyperplasia of the tissue between the glands, *Jones* believes in an excessive contraction of single bundles of the muscularis mucosa as a cause. Undoubtedly this gastritis polyposa

with its mammelonated appearances may be formed by a variety of very different processes.

INFLAMMATORY ATROPHY:

The progressive plastic character of the inflammation just depicted may lead to a retrograde metamorphosis before it has progressed very far, in some cases it may not develop at all but the disposition to break down and atrophy may start early in the disease. These atrophic changes are most marked in the glandular elements and may be limited to these. Sometimes the inflammations of the mucosa and gland cells have a degenerative tendency from the outset and no hypertrophy or hyperplasia enter into the anatomical picture. The surface columnar epithelium and the cylindrical epithelium of the vestibular alveoli falls prey to a mucoid degeneration and desquamation. The epithelial cells of the peptic glands undergo a fatty degeneration. During this atrophy the mucosa changes to a thin, smooth, pigmented or slate gray membrane.

This atrophy may be limited to the mucosa while at the same time hypertrophic processes go on unhindered in the submucosa and muscularis, then again the atrophy may extend to the latter layers and bring about a wasting of all gastric layers, this was formerly spoken of as *Taber of the Stomach*, the *Phthisis ventriculi* of Rokitsansky. Under these irreparable atrophic states anomalies in the gastric volume may develop but dilatation is more frequent here than contraction.

Atrophy of the stomach may occur without preceding chronic gastritis, it then takes the character of a simple degenerative process and follows severe anaemic and cachectic states and also grave infectious diseases and poisonings.

When confronted with cases of gastric atrophy with absence of hydrochloric acid the ferments and enzymes and coexistent anaemia it is sometimes very difficult to decide which is the primary causative disease. In these cases it is well to bear in mind that anaemias, even those of a grave pernicious character are much more often a consequence of, or rather secondary to atrophy of the gastric mucosa. Our countryman Austin Flint was the first to call attention to the relation between anaemia and atrophy of the gastric glands. In 1860, (Austin Flint, *American Medical Times*.) he expressed the opinion that some cases of obscure and profound anaemia

mia are dependent upon degeneration and atrophy of the glands of the stomach (further contributions of Flint to this subject are to be found in the *New York Medical Journal*, March 1871, and in his *Principles and Practice of Medicine*, p. 477, Phila., 1881).

Since Flint's publication, cases have been reported by Fenwick, (*The Lancet*, 1877, July, *et seq*) Quinke, (*Volkmann's Samml. Klin. Vortrage*, No. 100, case b.) Brabazo, (*British med. Jour.*, 1878, July 27) Nothnagel, (*Deutsch. Archiv f. Klin. med.*, Bd. 24, p. 353) Bartel's, (*Berlin Klin. Wochschr.* 1888, No. 3) Scheperlen, (*Nordis. med. Archiv*, 1879, Bd. xi, No. 3) Osler, (Atrophy of the stomach with the clinical features of progressive pernicious anaemia, *Am. Jour. med. Sciences*, 1886, No. 4). Rosenheim reported two cases which appeared to be pernicious anaemia (*Berlin Klin. Wochschr.* 1888, No. 51-52.).

Inasmuch, as these cases of atrophy of the gastric mucosa are accompanied by marked changes in the blood, signs of break down in the red blood corpuscles, increase in the white and formation of macrocytes and microcytes, the question may arise whether pernicious anaemia is really an independent disease or is the result of gastric atrophy (Ewald *l. c.*). Atrophy of the mucosa that is not secondary to well-known stomachs or general diseases but occurring as a primary disease has been claimed to exist by Fenwick *l. c.*

The impression might be gained from the statements of some writers, that the hypertrophic hyperplastic form of chronic gastritis may change into the atrophic form from its fully developed stage. This would mean the total disappearance of the papillary, poly-poid proliferations of the état mammeloné, because the mucosa of the atrophic form is very smooth. This, according to Orth (*l. c.* p. 710) is very improbable, for he is of the opinion that the atrophic form is developed uniformly by transformation of cellular interstitial tissue into contracting cicatricial tissue, bringing about thinning of the mucosa and degeneration of the glandular elements without the intervening of hyperplasia above referred to.

Ulcerative processes are said to occur (Ziegler *l. c.*) when in the course of the disease, intense (hemorrhagic) inflammation effects necrosis of the epithelium and submucosa and its subsequent sequestration. In this way the so-called catarrhal gastric ulcers and hemorrhagic erosions are formed, which may be associated with hem-

orrhage. Cruveilhier (*Anatomie Pathologique du corps Humaine*) terms it a follicular gastritis, in which ulcers are said to originate in the follicular glandular apparatus. The catarrhal gastric ulcers of chronic gastritis are mostly small, round or irregularly indented. They are supposed to heal and form flat, pigmented cicatrices. Förster asserts that they may lead to perforation. Orth *l.c.*, whose statements merit confidence because of the scientific conservatism of the man, is of the opinion that ulcerative processes in the course of chronic gastritis are very rare.

THE PATHOLOGICAL HISTOLOGY:

The minute anatomy of the process is that of a parenchymatous and interstitial inflammation, perhaps the most graphic description of it is given by Ewald *l.c.* (translated by Morris Manges, New York, 1892). The cells of the peptic ducts are described as being partly totally destroyed, partly granular and partly shriveled up. Differentiation between the central, chief or principal cells (Hauptzellen) and the parietal, border or oxyntic cells (Belegzellen) is impossible.

Osler and Henry *l.c.* (also in the *Principles and Practice of Med.* by Osler) describe a case in which the greater portion of the lining membrane of the stomach was converted into a perfectly smooth cuticular structure, showing no trace of glandular elements, but enormous hypertrophy of the muscularis mucosa and formation of cysts.

SYMPTOMATOLOGY:

As a general rule the outset of chronic gastritis can not be determined with certainty, because it develops very gradually and insidiously either as a continuation of acute gastritis and of other diseases or as an independent disease, and because the initial symptoms not being very pronounced are generally disregarded, only the sudden aggravation caused by dietetic errors and other injurious influences lead to the recognition that a serious disease is present. The clinical picture varies considerably, although the signs of a disturbed digestion as indicated by dyspepsia, absence of appetite, eructation, nausea, vomiting, pressure and fullness in the gastric region, repeat themselves in various cases, first one symptom and then another will press to the foreground or be absent entirely. Perhaps the most constant of the early symptoms is absence of appetite.

ABSENCE OF APPETITE — ANOREXIA:

Even in the less serious attacks, this as a rule is present and may be aggravated to a disgust for the customary diet.

TASTE:

I have rarely observed a case of chronic gastritis in which there was not present a pharyngitis, posterior nasal catarrh, laryngitis or one of the forms of stomatitis, one or other or several of these, usually the latter. This condition of the mouth renders taste perverted, pasty sometimes distinctly offensive, acid, bitter or metallic.

NAUSEA is an early symptom, generally preceding emesis; but it may exist by itself for many hours without emesis and may even occur on an empty stomach.

ERUCTATION is present at some time in all of the cases, the gases that are brought up are either air or carbon dioxide or hydrogen, in some rare cases inflammable gases, marsh gas CH_4 for instance have been eructated (Ewald — Rupstein).

Vomiting, though not so frequent as in acute gastritis, is nevertheless quite frequent. In the chronic gastritis of drinkers it is often a regular occurrence every morning and then known as the morning vomit or *vomitus matutinus*, which Frerichs used to explain by the swallowing of the secretions from the pharyngeal catarrh and saliva during the night. The morning vomit is usually alkaline, inverts starch to sugar and gives the red rhodankalium KCNS reaction with chloride of iron.

In beginning and in not very grave cases the ferments pepsin and rennin are yet to be detected, but in later stages only hydrochloric acid slightly in excess of the deficit will demonstrate the ferments; this really shows that the proenzymes, not the perfect ferments are present. Finally, pepsinogen and rennet zymogen are absent in very advanced forms, even the mucus will cease to be secreted; this is an indication of the complete atrophy of the mucosa.

THE TONGUE is very frequently coated with a grayish white deposit most mark on the back and root of the organ. The impressions of the teeth are retained by it. At the edges and apex the tongue appears in a deeper red color with swollen papillae. The coating may disappear toward evening to reappear in the morning. Hensch (Klinik der Unterleibs Krankheiten, Berlin, 1863, p.

382) holds that the appearance of the tongue is really not always a mirror of the stomach but that its condition is to be regarded simply as an index of the existing state of the oral mucus membrane. Certainly the tongue is much more frequently the first organ to become diseased of the two, as it is nearer the outer world and its infections than the stomach. Therefore it might be supposed that catarrhal states of the tongue, mouth and throat may occur more frequently as independent diseases not secondary to diseases involving the stomach, than as secondary diseases extending from an antecedent disease of the latter. Schech, (*Krankheiten d. Mundhöhle*,) in addition to malformations and inherited or acquired form defects of the mouth, describes sixteen distinct diseases of the human mouth not including neoplasm, tumors and nervous diseases; and Seifert (Penzoldt and Stintzing's *Handbuch der Speziell. Therapie*, Bd. iv.) describes 23 mouth diseases. In the primary form all of these arise in the mouth and occur as secondary forms in acute inflammatory conditions of the digestive tract, particularly after infectious diseases.

I have paid particular attention to the state of the tongue, oesophagus and stomach at autopsies, and also during a large number of analyses of stomach contents, and must admit that the condition of the tongue is one of the most variable signs in gastric symptomatology. The cases of manifest disease of the stomach where a primary disease of the mouth is out of the question are extremely rare. By a critical review of the etiology of gastric diseases, one can not fail to be impressed with the fact that the prominent causes can and most often do affect the mouth and the stomach alike. The gastric disorders in which the tongue is most frequently unaffected are those associated with little gastric sepsis, *i.e.* ulcer, hyperacidity and neurasthenia gastrica. Whereas in diseases associated with much gastric fermentation or histological changes in the mucosa that may extend to the mouth or involve it through circulatory or nervous channels, the tongue is most often affected, these diseases are gastritis, carcinoma and dilatation.

In reviewing the statements of most authors on the condition of the tongue one can notice a lack of clearness and precision which doubtless indicates that the relation between remote and local causes is not well understood concerning this matter. A systematic bac-

teriological and histological study of coated tongues is very much needed in association with gastric diseases. The attempt to establish a definite characteristic condition of the tongue for every gastric disease has apparently thus far failed. The extension of glossitis and stomatitis to the stomach is very intelligible by the deglutition of infective material. But the various forms of gastric diseases may also extend upwards either by eructations, or direct cellular continuity. Then again the oral and gastric cavities are in intimate correlation and may mutually affect each other through the vascular and complex nervous channels. Fleischer (*l.c.* p. 820) holds that the importance of the coating of the tongue as a sign of gastritis has been much overrated, and that the tongue may be clean notwithstanding very evident chronic gastritis and may be coated when this disease is absent. Nevertheless he considers the frequent coincidence of coated tongue and gastritis remarkable but attributes it to a concomitant stomatitis.

GENERAL NUTRITION:

Chronic gastritis of long standing left untreated will inevitably affect the general nutrition. As von Noorden repeatedly emphasized, most dyspeptics don't eat enough and in consequence of this emaciated to such a degree that even physicians suspect a grave underlying disease, tuberculosis or carcinoma, where there is only a chronic gastritis. The absence of appetite is most frequently caused by suppression of secretion of hydrochloric acid.

Feeling of pressure and fullness in the epigastric region is in many cases complained of and may be evident on awaking or develop after ingestion of food.

CONDITION OF GASTRIC CONTENTS—SECRETION:

The results of microscopical and chemical analysis of the test meals or of lavage water early in the morning before any food has been taken, will vary according to the particular kind of chronic gastritis and according to the present state of the disease. Boas recognizes with regard to these points four varieties, viz: (1) Gastritis Mucosa, (2) Acida, (3) Atrophicans, (4) Anacida.

1. GASTRITIS MUCOSA OR MUCIPARA. As was pointed out (*page 97, part I*) before when Rhinitis, Laryngitis, Pharyngitis and Bronchitis can be eliminated, then large quantities of mucus in the gastric contents as a rule speak for chronic gastritis mucosa. The cases

not forming much mucus are extremely rare. The mucus formation can be best estimated by washing out the fasting stomach. There should be no difficulty in distinguishing between the gastric mucus and that derived from the respiratory passages. The former is generally thin, clear, glassy, stringy and flowing. The latter is thick, opaque, yellowish gray and lumpy. In the washing from the fasting organ one frequently finds organic, structural form elements of the mucosa, that have been minutely described in the last chapter. If these bits of mucosa are found at repeated washings, showing these elements either in conglomeration or singly, there can be no doubt of the existence of glandular chronic gastritis. Frequently the morning contents of the fasting organ show numerous leucocytes. The contents should be drawn by expression if possible without using water. In gastritis chronica, mucipara the contents may show normal amounts of hydrochloric acid, they may be neutral or alkaline.

STATE OF THE SECRETION:

Up to recent investigations it was uniformly stated that absence or great diminution of hydrochloric acid was a constant symptom of chronic gastritis. Boas argues that there is a form of typical inflammation of the stomach termed by him.

2. GASTRITIS ACIDA in which there is a normal amount of acid or even superacidity present (Boas — Ueber Gastritis acida — *Mittheil a. d. Natur forscher versaml.*, Wien, 1894.).

Even the mucus from the fasting stomach may turn red congo paper blue. In gastritis subacida or anacida the free hydrochloric acid is reduced or entirely absent, but combined hydrochloric acid may be present still. The digestion of albumen, loss of secretion of pepsin discs or fibrin in the thermostat, is much retarded or may be wanting entirely showing the suppression of the secretion of pepsin.

Dissappearance of Rennin and its Zymogen goes on simultaneously with that of pepsin. In cases with loss of rennin the zymogen of this ferment must be tested for.

ATROPHIC GASTRITIS both free and combined hydrochloric acid are absent and the tests for enzymes and proenzymes are negative. Milk that is taken or poured into the stomach is returned mostly in an unchanged condition. Martius and Lüttke *l.c.*, von

Noorden and others take the position that absolute deficiency of pepsin and rennet is never seen. On the basis of a large clinical experience I am prepared to state that the end stages of atrophic gastritis give no evidence of ferments in gastric contents by any of the known tests. Nor would it be rational to suppose that hypertrophic gastritis in which the stomach is converted into a hyperplastic dense hard mass of muscle and connective tissue with no histological remnants of a glandular layer, there could be any possibility of the formation of enzymes.

4. GASTRITIS ANACIDA. In this subdivision free hydrochloric acid is diminished or entirely absent, but combined hydrochloric acid is still present. Egg albumen discs are not at all or but slowly digested in the filtrate even after hydrochloric acid. The difference between this form and the atrophic form is but one of degree as in the latter all secretion is lost completely.

AGE:

This is preeminently a disease affecting adults, for the young are not so liable to abuse their stomachs, to be exposed to the manifold factors composing the etiology and their restructure and compensatory powers are greater. The majority of the cases are over 40 years of age.

THE CONDITION OF THE BOWELS is most frequently one of constipation. The absence of the antiseptic action of hydrochloric acid favors intestinal fermentation, flatulence and metervism.

THE URINE is rich in urates and phosphates and gives a strong reaction for indican. The total acidity of the urine is reduced. The state of *general health* is a variable one, the body weight may be reduced or remain the same for years; there may be many changes of the general condition from good to bad and *vice versa*, but as the chronic inflammation progresses, symptoms of general discomfort and indisposition to bodily or mental exertion are marked.

DIAGNOSIS:

The determination of chronic gastritis is one of the most uncertain things in the whole domain of the clinical pathology of digestion. It requires careful study, not only to distinguish chronic gastritis from other diseases but to distinguish the simple, mucus and atrophic (and gastritis chronica of Boas) forms from each other.

As a rule the primary and secondary forms can be distinguished without much difficulty: Generally speaking the diagnosis of chronic gastritis can only be satisfactorily established after the possibility of the existence of other affections of the stomach have been excluded. This disease may strikingly resemble the clinical picture of the gastric neuroses of ulcer and even carcinoma. As dilatation is a very rare complication, it is not a confusing factor in diagnosis. One should not make the diagnosis definite at the first examination but reserve the opinion until the patient has been studied at 3-4 visits. It has in some cases taken me much longer than that to obtain satisfactory evidence of the disease.

The best evidence is obtained from repeated microscopical and chemical examination of the wash water and test meals. It will be necessary to dwell upon the differential diagnosis between chronic gastritis and the neuroses, ulcer and carcinoma. The neurosis may present all the symptoms of a chronic gastritis, particularly the absence of hydrochloric acid, but then after patient and repeated test meal analysis it will be found that the neurosis will some day show normal and even excess of hydrochloric acid. The thing to do then is to wait for this evidence.

The presence of much mucus, epithelial cells and leucocytes in the wash water from the jejune stomach, speaks for chronic gastritis.

The demonstration of the enzymes and proenzymes is very valuable, as a normal amount of rennet zymogen and pepsinogen when hydrochloric acid is absent, (Jaworski's method of pouring in decinormal hydrochloric acid should be used.) speak for a neurosis and against gastritis. Absence of enzymes with absence of hydrochloric acid does not easily occur in the neuroses. In the very beginning of chronic gastritis the enzymes may be present even in normal amount, but they disappear gradually as the disease progresses.

By the time the physician is consulted the enzymes are very much diminished or entirely absent, and according to Boas this is an indication of an inflammation of the mucosa and not a neurosis. The differential diagnosis between *idiopathic chronic gastritis and ulcer* is decided by the symptom of pain which is always present in ulcer and usually absent in gastritis chronica. The ulcer pain is

localized, well circumscribed, very intense, and occur at definite times after taking of food. Haematemesis, of course, points to ulcer. The vomit of ulcer shows hyperacidity which is rarely present in gastritis.

From *Carcinoma* the differentiation is very difficult when no palpable tumor can be detected; this is intelligible when one reflects that carcinoma is always complicated with chronic gastritis. If a pyloric carcinoma is present the symptoms of stenosis, motor insufficiency, and stagnation of food with large amounts of lactic acid force themselves to the front.

A carcinoma however seems to strike a stomach suddenly with very severe symptoms and general disturbances, pain, emaciation and vomiting, whereas chronic gastritis is characterized by slow increase of the gravity of symptoms with alternating improvements and aggravations. It is an important fact that the motility is not disturbed in chronic gastritis and therefore the jejune stomach rarely contains anything but mucus and isolated cells and leucocytes. But in carcinoma the peristalsis is seriously impeded from the onset and therefore there must be stagnation, retention, and acid fermentation.

These retained ingesta occur even where there is no stenosis of the pylorus, as a result of carcinomatous invasion of the muscularis. Gastrectasia is an exceedingly rare result of gastritis, it can only occur from hyperplastic thickening of the pylorus, a thing seldom reported in the literature of this subject. As stated before presence of marked amounts of lactic acid is not observed in gastritis but in carcinoma it is frequent. Organic acids are rare in the test meals of gastritis, in carcinoma there is as a rule an excess of lactic and fatty acids early in the disease (Boas *l. c.*).

PROGNOSIS:

Chronic gastritis is a tedious but not a very serious affection, as many cases recover under suitable treatment. The prognosis must vary with the stage of the disease as presented and the intelligence and will power of the patient. Patients who will learn how to avoid further detrimental influences and who have the determination to carry out the dietetic and hygienic management, will recover. In case of incorrigible eaters or drinkers who resume trespassing against their stomachs on the slightest improvement, permanent recovery is doubtful.

THE TREATMENT OF CHRONIC GASTRITIS.

PROPHYLACTIC TREATMENT:

The prevention of the development of the disease requires avoidance of the causes given under the heading of etiology of acute and chronic gastritis. Special attention should be directed to the avoidance of continued abuse of alcohol. Every acute gastritis be it an independent, idiopathic, affection or secondary to other diseases must be carefully treated in order to avoid its transition into the chronic form. Very accurate directions regarding diet and mode of life must be given to all sufferers from Liver, Lung, Heart and Kidney diseases also to diabetics in order that they may be saved from secondary gastritis, for disturbance of appetite and impairment of digestive powers must inevitably render the fundamental disease more serious.

The chief predisposing factor in chronic gastritis is passive congestion, the accumulation of injurious metabolic products and heart muscle and renal insufficiency. In such cases of threatened passive engorgement, digitalis should be used early. One need not fear the appetite disturbing effect of the medicine as this is usually transient and I can confirm Leube's statement that an improvement of the appetite and of nutrition in general is observed after treatment by digitalis. The passive engorgement of the mucosa is more harmful than the drug.

If it is noticed in several attacks that the gastric symptoms improve on digitalis it is expedient to give the remedy at the outset of the slightest disturbance of appetite, since our experience has taught us that this will unfailingly get worse, if the stomach rebels against the remedy give the infusion by an enema into a rectum previously cleaned by warm normal salt irrigation or give it hypodermically.

LAVAGE:

When it is no longer possible to remove the causes that led to a chronic gastritis we may yet be able to remove those that maintain or aggravate the malady. These are the accumulation of mucus and the mechanical as well as chemical irritation of the stagnating contents, particularly when atony and hypertrophic stenosis exist. To accomplish this, emetics are unpractical because they

rarely effect a thorough cleaning and may increase the inflammation by the convulsive contractions they cause and their direct irritation. If an emetic is absolutely not to be avoided apomorphine hypodermically is the safest. Purgatives are even more deleterious because they also increase the gastric irritation, can not be used habitually and hurry the decomposing masses into the intestines, thereby precipitating an involvement of this tract and the dangers of intestinal putrefaction and autointoxication.

Lavage is the only correct procedure in chronic gastritis whenever increase of mucus, absence of hydrochloric acid, decomposition and a protracted stomach digestion are evident. The mucus often adheres very tightly to the gastric walls, since it appears as a rule toward the close of the washing. Its evacuation is facilitated by allowing the water to run in under high pressure and directing the patient to change his position, *i. e.* lying on his back, rising, or turning on his side during the lavage or by employing gastric massage. In very troublesome lavage my double current stomach tube can be recommended (part I, page 81.).

The solution of the mucus is effectually accomplished by adding 1 tablespoonful of salt and two tablespoonsful of sodium bicarbonate or baborate to a litre of warm water. To disinfect the stomach after the removal of mucus and fermenting ingesta the following remedies are approved aids; Salicylic acid 1-000, thymol 0.5-1000. Boracic acid 10. to 1000, chloroform water 5 to 10 to 1000, shake the chloroform with the water and after settling pour off the water using only the latter. Hydrochloric acid 8.0 to 1000, resorcin sublimate 10.-1000, benzöl 5.0 to 1000, solutions must be prepared only immediately before washing.

The frequency of the lavage depends upon the state of the stomach; there may be cases that do not require it oftener than once in 2-3 days, others require it twice in 24 hours, as a rule once a day is sufficient. The time of the washing should be so selected that the exhausted stomach may enjoy the longest possible rest after it. For this purpose six o'clock in the evening is best adapted, as it is then about six hours after the main meal of the day and only very light diet is taken after the lavage and before bed time. In other cases this hour may be inconvenient and an early hour before breakfast must be chosen.

Washing out the stomach is advisable only when there is much formation of mucus and where there may be stagnation of food. In atrophic or chronic gastritis without much mucus, frequent lavage is not necessary. In these cases the stomach tube is recommended, not to remove fermenting ingesta or mucus, but to treat the mucosa directly, to stimulate its sluggish secretion if enzymes are still to be detected, by irrigating with decinormal solutions of hydrochloric acid; also common salt solutions are useful for this purpose (about one tablespoonful to the quart).

BALNEOLOGICAL. The use of the Alkaline Springs at Saratoga; Bethesda, Wisconsin; and those at Bedford, Pa. The waters of *Carlsbad, Germany* have a far famed repute in the treatment of this malady.

DISINFECTION of the mouth should be done three times daily by wrinsing, brushing teeth and tongue with the following:

R

Acid Thymol	0.25
Acid Benzoic	3.0
Tinct. Eucalypt.	15.0
Alcohol	100.0
Ol. Menth. pip.	0.75

Pour enough into $\bar{\text{z}}$ iii of water to make it slightly turbid.

M.

If much mouth putrefaction is present, 0.8 HgCl₂ Hydrag. corros. bichlor. can be added, but patient must be warned not to swallow any of the liquid.

ELECTRICITY. — Both the galvanic and the faradic current should be used; the positive pole in the stomach with the Einhorn intragastric electrode, and, the broad plate electrode with the negative pole should be on the cervical spinal column.

DIET.— In patients that eat very little, the appetite should first be aroused by giving the following:

R	Acid hydrochloric dil.	fl. ʒiv	
	Strychnin Sulphatis	gr. ⅓	
	Elixir Gentian	g. s. ad. fl. ʒvi	M.
Sig.	fl. ʒss in ʒii Aquæ after meals. t. i. d. through a glass tube.			

In the subjoined diet list which I have suggested for my Baltimore patients, and, which is used at the private sanitarium for digestive diseases, will be found a *Menu* which will be more adapted to the market and palate of our American people.

HEMMETER'S DIET LIST FOR CHRONIC GASTRITIS.

At 7.30 A.M.

If bowels are regular, ½ pint of hot normal saline solution.

If bowels are constipated, a pint of cold Saratoga Vichy or plain cold water.

Breakfast, 8 A.M.

100 grms. or ʒiii ⅓ Farina boiled with milk = 127 Calories.

or 100 grms. Cerealine " " "

or 100 grms. Breakfast Wheat (strained) "

One soft boiled egg 80 "

Two ounces wheat bread, toasted 156 "

One ounce of best fresh butter 212 "

One cup of wheat coffee, (made of 100 grms. of coffee made from roasted choice wheat and 150 grms. of milk). Instead of this the same portions of tea and milk or cocoa can be used = 100 cal.

Sugar, 10 grms. (ʒii ss) 40 "

The farina or cerealine will taste better if eaten with a roasted apple.

As the digestive power improves, the egg is presented in form of omelette or poached, on toast.

10.30 A.M.

100 grms. Scraped Ham ($\bar{3}iii\frac{1}{3}$)	120	Calories.
30 grms. Crackers or toast ($\bar{3}i$)	107	"
226 grms. or 8 ounces of Broth	306	"

Instead of broth — milk, kefir and matzoon may be permitted in the same quantity.

Dinner, 1 P.M.

Soup made of 250 grms. or $\bar{3}viii$ bouillon, 30 grms. or $\bar{3}i$ of rice or tapioca, 10 grms. or $\bar{3}ii$ ss of butter and 1 egg = 282 cal.

In case of much weakness and emaciation, $\frac{1}{2}$ tablespoonful of *somatose* should be added.

The patient must not be aware of the addition of artificial foods.

120 grms. of breast meat of broiled fowl, = 288 Calories.

or scraped tenderloin formed into patties and broiled,

or steamed or broiled bluefish, trout, white or yellow perch,

or broiled rock fish, or sweet breads.

50 grms. or $\bar{3}ii$ potatoe purée = 637 calories.

100 grms. or $\bar{3}iii\frac{1}{3}$ carrots, steamed 40 "

or 100 grms. purée of beans or peas,

or 100 grms. strained tomatoe purée,

100 grms. finely divided spinach,

1 cup custard made of 2 eggs, 160 "

Or instead of this, 100 grs. of sherry gelatine, or stewed apples, or plums, or rice in form of very light rice pudding made with slices of apple, no raisins.

One glass of (100 grms. — $\bar{3}iii\frac{1}{3}$) Hungarian Tokay, = 50 cal.

Instead of the meats given, the patient may, for a change, be allowed broiled pigeon or venison which must not be gamy; also meat dumplings of scraped beef, scraped pork made into balls with bread crumbs, zwieback crumbs, egg and butter, cooked in bouillon and a separate sauce is made and flavored with scraped sardelles.

At 3 P.M.

1 cup of chocolate made with 30 grms. or $\bar{3}i$ of breakfast cocoa or v. Mehrling's *Kraft Chocolate* and $\frac{1}{2}$ pt. milk, = 135.5 Cal.
30 grms. crackers, coffee cake without grated nuts, cinnamon short cake with but the faintest trace of cinnamon, = 107 cal.

If the sweet chocolate is not agreeable, plain milk or a glass of light rhine wine with crackers are allowable. Coffee in small quantities may be added to the milk at this hour.

Supper, 6.30 P.M.

Broiled, panned or raw oysters, 240 grms. or ㉟viii , = 70 cal.

If there is sub or anacidity, a little grated horse raddish, lemon juice or cutsup to the raw oysters should not be forbidden.

Crackers, ㉟ii or 60 grms. = 107 calories.

Butter, ㉟i or 30 grms. = 212 “

$\frac{1}{2}$ pint of reliable rhine wine, = 50 “

or $\frac{1}{2}$ pint of imported beer, $\frac{1}{2}$ pint of tea and milk.

Instead of the oysters, — little neck clams, fresh scraped beef, finely cut roast lamb or beef, cold, smoked chipped beef or smoked tongue will answer.

ULCER OF THE STOMACH.

ULCUS VENTRICULI PEPTICUM, ROTUNDUM, PERFORANS,
RODENS, CORROSIVUM, E DIGESTIONE.

ULCER OF THE stomach is a loss of substance of the gastric mucosa characterized by very little tendency toward healing, but rather by destructive progression, both in a longitudinal direction, in a line with the internal surface as well as toward the depth of the mucosa. It may occur in two forms; (1) the acute and (2) the chronic.

The acute form extends so rapidly from the mucosa toward the peritoneum with such little lateral involvement that Rokitansky's original comparison, "*As if the ulcer were cut out with a punch,*" has become the classical expression of the text books.

In the chronic form the destructive process is not so rapid; it extends more laterally, producing a terraced or shelving appearance of the edges and sides, so that it may be funnel shaped.

Perforation into an artery vein or into the peritoneal cavity occurs in both forms. The chronic form has a tendency to healing, but in so doing causes cicatricial contractions and deformity. The acute form may terminate in healing, but owing to its limited lateral extent, the small cicatrix rarely causes deformity.

It is very probable that the acute ulcers have a different etiology (corrosive, toxic action — trauma by sharp, hard materials in the food) from the chronic eroding type, to which the following description appertains more especially.

SELF DIGESTION OF THE STOMACH — GASTROMALACIA.

If an animal is killed while in full digestion, the stomach may undergo self digestion providing the body is kept warm. In human beings that died suddenly, whilst the gastric digestion was at its height, it was found at the autopsy that not only the stomach had been digested, but also the spleen, and, that this process had extended through the diaphragm into the lungs.

The question naturally arises: What protects the stomach from autodigestion from its own secretions under normal conditions? This is an inquiry that concerns the fundamental properties of living matter, for it includes the non-digestion of the intestinal tract by the alkaline pancreatic juice and succus entericus, the same property as observed in the digestive tracts of invertebrates and even in the unicellular organisms, the amæbæ and plasmodia of mycetozoa for instance, in whom Metschnikoff, le'Dantec, Greenwood, Saunders and myself have shown that a secretion is formed in the digestive vacuoles of the unicellular protoplasm which digests foreign proteid material but not the living substance of the cell itself. (See—On the Role of Acid in the Digestion of Certain Rhizopods, by J. C. Hemmeter, Phil. D., etc., in *The American Naturalist*, Aug., 1896, p. 619).

Riegel asserts that in consequence of and by means of hyperacidity an ulcer may develop from an erosion or injury of the mucosa, which, though seemingly unimportant in itself is retarded in its healing by autodigestion. Of course it is intelligible that the hyperacidity (as Ewald, Ritter and Hirsch point out) may be the result just as well as the primary cause of ulcer.

Fleischer points out that the hyperacidity may change normal clonic contractions of the gastric muscularis to intense tonic spasms that last an unnaturally long time and produce local ischæmiæ and impediments to the exit of venous blood. This, if it occurs, must inevitably be followed by local disturbances in nutrition of the

mucosa and eventually hemorrhages of a circumscribed character, out of which the autodigestive process readily forms erosions and ulcers.

ETIOLOGY.

The deductions from the preceeding summary of experiments and observations are above all (the establishing of) four principal factors in the etiology of ulcer.

1. An impaired vitality or resistance of portions of the mucosa.
2. Hyperacidity or supersecretion.
3. An altered state of the blood.
4. Local bacterial infection.

There are a number of well authenticated cases on record, proving that direct trauma may cause gastric ulcer. (Vide Einhorn, *l.c.* p. 191, also others reported by C. Hoffmann, Leube and Eichhorst).

According to Sidney Martin (*l.c.* p. 410), there are three common causes of the death of the tissue which precedes ulceration.

MECHANICAL AND CHEMICAL CAUSES:—

Ingested fish bones, egg and oyster shells, seeds and corrosive poisons by directly destroying the tissue lead to ulceration; and an injury to the mucous membrane, which is subsequently exposed to the continued action of an irritant, will also lead to an ulcer.

INTERFERENCE WITH THE VITALITY OF THE TISSUE:—

The vitality of a particular part of the mucous membrane may be diminished by local and chronic disease or by interference with the circulation over a certain area. This latter usually occurs by the means of thrombosis or embolism. Thrombosis takes place in connection with disease of the vessels and in association with a diminished quality of the blood and a slowing of the local circulation; embolism may be infective or non-infective and is usually capillary.

BACTERIAL INFECTION:—

The infective processes of the digestive mucosa with which we are most familiar are the ulcerative processes of typhoid fever, certain dysenteries and tuberculosis. In the gastric ulcer however there is another kind of bacterial infection, which is not accom-

panied with the signs of active inflammation and is termed by some authors, *bacterial necrosis*.

The process is characterized by the invasion of bacteria usually in the lower depths of the mucous membrane by their growth and subsequent necrosis of the tissue. Although the secretion of hydrochloric acid is germicidal to many bacteria it must be remembered that the spores are not destroyed by it and that the invasion may take place during the periods of rest of the glands in the intervals of digestion when no hydrochloric acid or very little is secreted.

There is room for the suggestion that the primary necrosis is due to bacteria and the ensuing ulceration caused by the action of the gastric juice. The bacteria can exist in the cells around and beneath the floor of the ulcer notwithstanding a very high degree of hyperacidity.

In a number of cases which I examined by the most approved cellular and bacterial stains, the bacteria were present throughout the layers even in the peritoneum, whilst the floor of the ulcer was in the muscularis. It is conceivable that they pave the way for autodigestion by causing necrobiosis of the tissues in which they are imbedded. No bacterium was so far obtained in pure culture, but the one most frequently observed was a bacillus very much resembling that of anthrax, and the Oppler—Boas bacillus.

THERMIC CAUSES are the ingestion of very hot food and drink taken when the organ is empty.

CUTANEOUS BURNS are in some very curious causative relation to gastric and duodenal ulcers. The last two factors, hot food and large cutaneous burns explain the rather frequent occurrence of gastric ulcer among cooks; who are in the habit of tasting foods that are still on the fire and more liable to skin burns.

CONSTITUTIONAL CAUSES are generally brought about by such diseases as effect alterations and degeneration either in the composition of the blood or in the vessels. These are chlorosis, anaemia syphilis, tuberculosis arteriosclerosis, fatty, amyloid and aneurysmal degenerations of the arteries, thrombi, emboli, trichinosis and malaria.

The following table is given by Welch, representing the age in 607 cases of open ulcer collected from hospital statistics. (*Peper's system Medicine*, vol. ii).

Age	1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	90-100	over 100
No. of Cases.	1	32	119	107	114	108	84	35	6	0	1
Totals.	33		226		222		119		7		

From this table it is apparent that the largest number of cases is found between 20 and 30. Three fourths of the cases occur between 20 and 60.

DIAGNOSIS:—

The accompanying table shows the cardinal points in diagnosis, of which there are really very few. The most important are haematemesis with perhaps a preceeding severe gastralgia, the chronicity of the process and the characteristic pain points *i.e.* the epigastric and the dorsal. The epigastric painful region is dependent in its limits upon the location and position of the stomach, if the latter is normal the pain point will be close to the xyphoid cartilage, but if the stomach is low down the point may be 2-3 inches lower. The dorsal pain area is present in about $\frac{1}{3}$ of the cases (point rhachidien of Cruveilhier) and is located at about the level of the tenth or twelfth thoracic vertebra and has a lateral extent of 2-4 cm. and a vertical extension of 1-4 cm, in the majority of instances it is present only on the left side, though it is observed at times both on right and left and in rare cases only on the right side. A chronic process must be assumed where temporary and even quite long periods of relatively good health intervene between attacks of pain.

Wherever a chronic morbid process can be determined upon with accuracy and the characteristic pain points are present at the same time, the diagnosis according to Boas should be certain (*l.c.* p. 41). He attributes less importance to analysis of the gastric contents. There are however atypical forms which present some difficulty in diagnosis. Thus there are cases rarely observed in which the patients never complain of pain, nor has the food any distressing effect upon the stomach. In other cases although pain is pres-

Table of Differential Diagnostic Points.

	GASTRIC ULCER.	NERVOUS GASTRALGIA.	HYPER- CHLORHYDRIA.	CANCER.
Age.	Rare in youth, frequently increasing progressively from puberty to a very advanced age.	Most frequent between the ages of 18 to 35.	Met with in all periods of life, except in youth, when it is quite rare.	Middle age and advanced life.
Sex.	More frequent in women. (2:1).	More frequent in women.	More frequent in men.	No marked difference between the two sexes.
Epigastric pain.	Quite intense, appears shortly after meals, grows severer on pressure; disappears at the end of the digesting period, seldom perfectly free periods.	The pain appears without regularity and is not in any way dependent upon the meals; is relieved by pressure and shows intervals of several days duration which are perfectly free from pain.	The pain appears about two or three hours after meals and disappears after partaking of some food especially meat, milk, eggs or after the administration of bicarbonate of sodium.	The pain is less intense in character but more steady; they are seldom free intermissions during which no distress is felt in gastric region.
Appetite.	Appetite not impaired although patients as a rule eat less on account of their suffering.	Variable.	Often increased.	Appetite as a rule very poor.
Tongue.	Dry and red, showing a white stripe in the middle, or smooth and moist or slightly furred.	Presents a normal appearance.	Is either clean or slightly furred.	Almost always thickly coated.
Taste.	Nothing abnormal.	do.	do.	Very often bitter or sour.
Belching.	As a rule absent; if present without any bad odor.	do.	do.	As a rule present and very often associated with a disagreeable, even fetid odor.
Regurgitation.	At times present frequently water brash associated with pyrosis.	Not present.	Water brash and pyrosis quite frequent.	No water brash; pyrosis quite intense.
Vomiting.	Appears in some cases soon after meals.	Shows no regularity in its appearance.	No vomiting.	The vomiting as a rule, occurs not after meals but once or twice a day or once in two days, the quantity being often very large.
Haematemesis.	Vomiting of a large quantity of blood, either clear red or of coffee-ground color. Blood is also found in the stools. A repetition of the haematemesis may occur on the following day, but if once arrested it does not reappear for quite a long period.	No vomiting of blood.	No vomiting of blood.	Vomiting of blood occurs; the quantity is relatively small, the color ordinarily coffee brown. The blood appears in a decomposed condition; frequently a fetid odor. The vomiting often recurs with short intermissions.
Secretory function.	1. Gastric juice as a rule increased. 2. Lactic acid absent.	Variable. Absent.	Increased. Absent.	As a rule highly decreased. As a rule excessive.
Tumor.	No tumor, rarely however if the ulcer is near the pylorus the latter becomes thickened and can be felt as a smooth lengthy body.	No tumor.	No tumor.	Tumor very frequently palpable; presenting, as a rule, an uneven surface; is painful to pressure and easily movable.
Perforation.	Perforation might take place after a short period of illness.	No perforation.	No perforation.	Perforation occurs only in the last stages of the disease.
Complexion.	Complexion commonly fresh but anaemic after severe losses of blood.	Complexion Pale.	Complexion Pale.	Complexion sallow and yellowish; skin dry; marked cachexia.

ent, it is not aggravated by taking food. In some well diagnosticated cases, food of all kinds was well borne. In all of these well authenticated forms, the diagnosis was assured by characteristic unmistakable symptoms such as hæmatemesis and hyperacidity coming on afterwards.

Concerning hæmatemesis it should be said that the differentiation of pulmonary from gastric hemorrhage may become necessary. The differentiation may be facilitated by a study of the subjoined schema.

HEMORRHAGE FROM

LUNG.

1. Blood is bright red, foamy.
2. Previous history or cardiac disease.
3. Physical signs point to a pulmonary or cardiac affection—the stomach may be affected secondarily.
4. Pulmonary hemorrhage is followed by rusty colored sputa for days (generally) but there is no blood in the stools.

STOMACH.

1. Blood is dark brown, partly coagulated — frequently mixed with food.
2. Previous history points to a gastric affection or stasis in the portal system.
3. Physical examination evinces a gastric or hepatic affection or stasis in portal circulation.
4. Gastric hemorrhages are frequently associated with tar colored stools.

The diagnosis becomes complete if the characteristic pain points are present with prompt aggravation of pain soon after taking food. Vomiting showing hyperacidity, hæmatemesis and a history of chronic trouble.

The blood coming from the stomach does not necessarily originate from an ulcer. One may in rare instances be called upon to exclude carcinoma, portal vein stasis producing passive congestion, gastric varicosities, toxic corrosions, traumatisms scurvy, acute yellow atrophy of the liver. The hemorrhages of carcinoma are small in quantity compared to those of ulcer, and in cancer the blood is more frequently decomposed and of a coffee or chocolate brown color and there are rarely any bloody stools. Charcot has reported hæmatemesis in hysteria, [crises gastriques,] and Debove

suggests (*l.c.*) that organic and functional nervous diseases may be coincident with ulcer. In sudden gastric hemorrhages the previous history will as a rule enable one to distinguish between the above mentioned possibilities. In hemorrhage from passive congestion due to stasis of the portal vein the epigastric pain is very slight or absent entirely.

CHOLELITHIASIS may be confounded with ulcer when there has been no blood in the vomit or stools nor any grit sand or stones in the evacuations. The following signs and symptoms are then of value. The pain in hypatic colic is not in connection with the taking in of food and it draws from the median line to the *right*. The dorsal pain point of ulcer is located at the level of the twelfth thoracic vertebra, to the *left* and very close to the body of the twelfth vertebra. But the dorsal pain point of cholelithiasis is located to the *right* about 2-3 fingers breadth from the twelfth dorsal or first lumbar vertebra. In ulcer there is very rarely any pain on the *right* side but even if there is, it is much less intense, and in cholelithiasis there is rarely any pain to the *left* of the spinal column.

In cholelithiasis the right lobe of the liver is enlarged after an attack, the gall bladder also (if palpable) and during the interval between the attacks all kinds of food can be eaten with impunity. The amount of hydrochloric acid in gastric contents is normal or subnormal in ulcer-hyperacidity. Icterus when repeatedly observed following attacks of pain, strengthens the diagnosis of cholelithiasis, but it must be emphasized that with duodenal ulcer icterus is not rarely observed. In private practice I have observed a case in which cholelithiasis and gastric ulcer occurred contemporaneously.

DIAGNOSIS OF THE COMPLICATIONS AND CONSEQUENCES OF GASTRIC ULCER.

These are: — (1) The perforation Peritonitis, (2) Cicatricial Stenosis of the pylorus, (3) The transition of ulcer into Carcinoma or ulcus Carcinomatosum, (4) Hour glass stomach from cicatricial contractions, (5) Subphrenic Abscess, (6) Progressive pernicious Anaemia.

The diagnostic signs of perforative peritonitis are, (*a*) great rigidity of the abdominal muscles flat abdomen, (*b*) Disappearance

or diminution of Liver dullness which may be absent if only liquid gastric contents, no air escapes into peritoneum, (c) Vomiting.

According to Rosenheim, (*Zeitschr. f. Klin. med.* b. 17, s. 116) about 5 – 6% of gastric ulcers develop carcinoma at their margins and these carcinoma are said to be associated with a pronounced hyperacidity.

The so-called *hour glass stomach* may be produced by one or more cicatrices in the neighborhood of the Antrum Pylori. Cicatrices of the duodenum may cause a dilatation beyond the pylorus thus the latter will constitute the narrowing or isthmus of what very much resembles an hour glass stomach (Reiche — *Talub d. Hamburg. Staatskrankenanstalt*, 1890, p. 180).

SUBPHRENIC ABSCESS—PYOPNEUMOTHORAX SUBPHRENICUS.

In 1880 Leyden first described a combination of diseases which followed perforative peritonitis or escape of pus from the intestines into the peritoneum. A purulent exudate forms in the lower parts of the right or left thoracic cavity under symptoms of inflammation, but no coughing or expectoration is connected therewith. The posterior and lower thoracic region gives dullness on percussion, absence of vesicular murmur and fremitus. Metallic sounds can be made out when one percusses and auscults simultaneously. The succussion sound is distinct. The lung is distinctly intact above these parts. The respiratory murmur is vesicular and the fremitus maintained down to the fourth or fifth rib; from here on the respiratory murmur suddenly ceases.

The dullness that corresponds to the exudate changes with various positions of the body. The signs of equally distributed pressure in the pleura are wanting. The movements of the corresponding half of the thorax are not coordinated, the intercostal spaces almost wiped out, the heart slightly pushed to the other side.

If the exudate is on the right side, the liver projects far into the abdomen and can be felt at or below the umbilicus. The exudate may perforate into the respiratory passages and cause sudden abundant expectoration of foamy pus. In 1894, Karl Madl collected 179 cases of subphrenic accumulations of pus. In 20 % of these cases, of perforating ulcers of the stomach or duodenum were found to be the causes. Progressive pernicious anaemia as a concomitant phenomenon of ulcer can be recognized by the reduction of the number of red corpuscles, the appearance of poikilocytes, microcytes and macrocytes.

TREATMENT OF GASTRIC ULCER.

PROPHYLACTIC:—If gastralgias are frequent in a person afflicted with hyperacidity, the diet must be very mild and unirritating, a milk diet will be the safest, sudden deviations in the temperature of the food must be avoided and daily evacuations must be effected by suitable diet.

The dietetic and medicinal treatment will vary according to the presence or absence of hematemesis. (Treatment of hematemesis and the period immediately following it). During the stage of blood vomiting the patient must remain absolutely quiet in bed and not even arise for urination or defecation. Positively nothing should be permitted by the mouth not even ice.

If the patient is well nourished no alimentation by the rectum is advisable because this necessarily disturbs the rest and compels the stomach to move because of the changes in position required. If the patient is weak and anaemic, a nourishing enema may be imperatively indicated every 4 hours. The enema most favored is that of Boas consisting of 250 Gr of milk, the yolks of two eggs, a teaspoonful of salt, one ounce of good claret and 1 tablespoonful of aleuronat flour. Previously to giving an enema for nutritive purposes the rectum and colon must be cleansed by a high irrigation with one liter of warm water. The above mixture is thoroughly mixed by means of an egg beater warmed to about 99° F. and permitted to run in under gentle pressure, care being taken that the tube is introduced as far up into the sigmoid as possible.

When the hematemesis is copious and persistent a hypodermic injection of Ergotole 20–30 minims should be given at once. With this preparation of Ergot I have had extensive experimental and clinical experience (see *Medical News* for Jan 31, Feb. 7, Mar. 7 and 14, 1891.—An experimental and clinical study of Ergot by J. C. Hemmeter). At the same time an ice bag is placed over the epigastrium and if pain is severe an injection of $\frac{1}{4}$ Grain of Morphine should not be delayed as by the ease and quiet it brings about this drug acts adjuvant to the hemostatic. For three days following hematemesis this treatment should not be changed and no food allowed by mouth. The treatment from the fourth to seventh day after, consists of absolute rest in bed, a wet pack covered with oiled silk and bandage applied to the epigastrium.

And now one may resume feeding by the mouth but in form of liquids only. Half milk half lime water, or milk with a small addition of coffee or tea never more than lukewarm. Also Beef tea to which, lactose, meat powder or somatose have been added and egg albumen water. Chocolate, yolks of egg and all alcoholic beverages must be forbidden in this stage.

In the second week after the hemorrhage a typical cure for ulcer according to principles laid down by Wilson Fox, (*Diseases of the Stomach*, 1872, p. 146) v. Leube (*Ziemssen Handbuch*, vii, 2, p. 120) and v. Ziemssen (*Volkmann's Sammlung Klin. Vortr.*, No. 70). These systematic treatments are in the main, rest cures combined with the daily use of a glass of Carlsbad Mühlbrunnen water, liquid or semiliquid diet and hot applications to the epigastrium. Every morning the patient takes a glass of (40° R) warm Mühlbrunnen in which 5–10 grams of natural or artificial Carlsbad salts have been dissolved, spongiopiline cut any requisite shape and dipped into hot water is applied externally to epigastrium and renewed every 3 hours night and day. The diet consists mainly of milk and whipped eggs, if there is great weakness the above enemata containing perhaps 3ii claret should be given, and if the pulse is feeble hypodermic injections of digitaline gr. 1–30 and strychnia gr. 1–30. In one case of profuse hæmatemesis, I gave an intravenous injection of 500 c.c. of sterilized normal salt solution. The pulse had left the wrist and was barely perceptible at the carotid, the effect was prompt and the opinion of the assisting colleagues was that life was saved thereby, the case recovering later on under the nitrate of silver treatment.

In the *third week* when the pain in the epigastrium and general cardialgia have ceased, the patient may be permitted to rest on the sofa and the Carlsbad water is continued. I might remark here that the Saratoga Carlsbad acts quite as well as the imported. In fact the sole object of the Carlsbad water in the cures of Leube and Ziemssen is the neutralization of the hyperacidity and the promotion of intestinal evacuation. One must not gain the impression that Carlsbad waters or salts have any direct or specific curative effect. Ewald (*l.c.* p. 275) declares that many a patient who went to Carlsbad might have recovered more rapidly if he had taken the rest cure at home. To neutralize the hyperacidity and prevent its autodigestive action.

I usually give the following:—

R	Magnesiae ustae		
	Sodii Carbonatis		
	Potassii Carbonatis	a.a. 5.0 (3i + Gr. xv.)	
	Sacchar lactis	25.0 (3vi + Gr. xv.)	
Sig.			M.

Half a tablespoonful dry on the tongue every 3 hours.

In the third week one may permit dipped cakes, toast or zwieback. Broiled sweet bread or calfs brain, dumplings made of finely divided meat, broiled pike, blue fish, trout, oysters in very small quantities. In the *fourth week* purées made of potatoes peas or beans, rubbed through a sieve, stewed apples, pears and plums. A glass of approved claret mixed with Saratoga, vichy may be allowed. All vegetables that can be made into purée (gruel) from such as spinach, carrots, peas, etc., etc. For many years the patient must avoid raw fruits, all sour, acid or spiced food and drink, ice cream and all cold and hot beverages. If there has been no haematemesis the treatment had best be carried out along these lines also. In rebellious cases of recurrent gastralgias, vomiting and hyperacidity, McCall, Anderson (*Brit. Med. Journal*, 1890, May 10) and H. B. Donkin (*the Lancet*, 1890, Sep. 27) recommend a total *abstinence* cure of 2 to 3 weeks, during which the patients are fed exclusively by rectal enemata (3 to 4 in the day). Hot applications to epigastrium are also used. After 10 days of rectal feeding they cautiously and slowly return to feeding by the mouth (milk buillon, egg albumen). I have tried this in a number of cases in whom relapses had occurred after the rest cure and can speak in favor of the method. Gerhardt and Boas speak very favorably of nitrate of silver in light cases of gastric ulcer. The latter begins with R Argenti Nitratis 0.25 to 120, peppermint water—one tablespoonful 3 times a day on an empty stomach—then the dose is increased to 0.3 to 120 of water of which two bottles are taken and finally 0.4 to 120 water of which also two bottles are advised. This is combined with a sparing diet and as much rest as possible.

Fleiner and Kussmaul recommend Bismuth subnitrate in all irritative conditions of the gastric mucosa—old ulcers, erosions, excoriating carcinomata. Fleiner employs it in the following manner;—10 to 20 grams (150 to 300 grains) of Bismuth subnitrate are

stirred in 200 c.c. of warm water. After the stomach has been thoroughly cleansed by lavage, this suspension is poured into the stomach and allowed to remain 3 minutes, then the clear water is siphoned, out the Bismuth remaining behind and forming a coating to the injured places in the stomach — it is a modified direct or local treatment. I usually employ five drachms of Bismuth subnitrate and one drachm of Bismuth subgallate in a pint of warm water—having previously thoroughly cleansed the stomach with solutions of sodium bicarbonate (3ss to pint). In cases in which Fleiner's treatment can be employed it relieves pain promptly, reduces the hyperacidity and promotes healing, it is worth trying. The anaemia following ulcer may require iron arsenic, strychnin. Iron preparations must contain no acid.

SURGICAL TREATMENT becomes necessary when after a trial of aforesaid methods the ulcer or ulcers prove very obstinate and not amenable to medical treatment because hemorrhage may become so abundant and frequent as to endanger life, or lastly, because of perforation. Nelson C. Dobson, (*Bristol Medical Surg. Jourl.* 1883) first advocated surgical interference for perforating gastric ulcer. In this country Robert F Weir of New York has contributed the most important work to this domain of surgery.

His last important paper (Rob. F. Weir and E. M. Foote—The Surgical Treatment of round ulcer of the stomach and its sequelae, etc., *Medical News*, April 25 and May 2, '96) contains an account of seventy-two cases of laparotomy for acute perforation of gastric ulcer. For particulars concerning the surgical treatment of gastric ulcer the reader is referred to textbooks on abdominal surgery and the publications of Weir. Gastric ulcers have recently been excised entirely, the sequelae thereof have been removed by severing of peritonitic adhesions and hour glass stomach much improved by gastro anastomosis (see v. Hacker — Ueber Magenoperationen bei carcinom u. b. narbigen stenosen, published by Wilh. Braumüller—Wien and Leipzig 1895).

CARCINOMA VENTRICULI.

CANCER OF THE STOMACH.

GASTRIC CANCER is characterised by the progressive cachexia which distinguishes it from all other chronic affections of the stomach. It is not sufficient for diagnosis and therapy to diagnose the simple presence of cancer, since these neoplasms vary so greatly; according to their location at the cardia, the curvatures and the pylorus and in so many important symptomatic and diagnostic points, that they compel separate consideration. It is expedient to consider these malignant tumors under three headings, vid:—

(1) Carcinoma of the cardia, (2) of the body of the stomach, *i. e.* the curvatures and the fundus and walls, (3) of the pylorus.

CARCINOMA OF THE CARDIA: — (a) Signs and symptoms, complaints of an uncomfortable feeling of a foreign body and of pressure above the gastric region, particularly after the ingestion of food. Sensations of pain are not contemporaneous with swallowing of food but occur independantly. On ingestion of food a sensation as if the same becomes clogged or is caught before it reaches the stomach; patients imagine that copious draughts of water gives relief, most likely because this can pass through the stenosis caused by the neoplasm. Another important symptom is vomiting, which is not actual gastric vomit, but the wrenching up of mucus and few food particles. The cause of these regurgitations of masses of mu-

cus is the formation of a large dilatation of the œsophagus above the stenotic cardia carcinoma. In this œsophageal diverticulum or dilatation, the food is caught, retained, putrefies and is eventually vomited up again. There is also a catarrhal œsophagitis present at this place. Liquid or semi-liquid substances may for a long time be able to pass, while relatively solid substances give rise to the difficulties stated. Later on, as the stenosis increases, liquids can not pass either, and loss of appetite and strength goes on uninterruptedly.

If an obstacle to the passage of the sound can be ascertained at the entrance to the stomach in a person over 30 years of age the diagnosis of cancer of the cardia becomes certain. Under all such suspected cases only a soft elastic tube should be used for explorative sounding. In two cases in private practice during the last three years I was enabled to establish the diagnosis by microscopical examination of small portions of the carcinoma that were brought up with the sound. These neoplastic fragments are occasionally found in the eye of a lower opening of the sound and they constitute a definite criterion. In one of the above cases, the diagnosis was confirmed by my colleague Dr. Einhorn and in the other by autopsy. In addition to the sounding and the cancerous fragments the following signs are of diagnostic importance:—

1. Percussion of the region over xiphoid cartilage is very painful.

2. On the sound blood will frequently be found mixed with the extremely foetid mucus, and at times nests of cancer cells.

3. On placing a stethoscope over the epigastrium, normally two deglutition sounds can be heard. One is synchronous with the beginning of the act of swallowing and the other is heard from seven to twelve seconds later. Now in carcinoma of the cardia, the second deglutition sound which signifies the entrance of liquid into the stomach may be much delayed or absent entirely, this sign is of importance *per se*.

4. Supraclavicular swelling of the lymph glands, if palpable, support the diagnosis also.

5. Lauenstein asserts that there is a systolic murmur audible in the epigastrium, due to pressure of the tumor upon the aorta. According to Boas this is an inconstant sign.

Duration of the disease is 6 to 9 months after the first symptoms are manifested, death occurs as a result of gradual exhaustion, marasmus, aspiration pneumonia, secondary carcinomata in the liver and other organs and intercurrent hemorrhages.

DIFFERENTIAL DIAGNOSIS from chronic gastritis is difficult in the beginning of the cancer, as in both the presence or absence of hydrochloric acid is no criterion, but as the cancer progresses, the sound will settle the doubt in locating the stenosis. From œsophageal ulcer, the cardia carcinoma is differentiated by the fact that pain is immediately associated with deglutition of food, by the age of patient, (see tables of ages at which ulcer and cancer are most frequent) by the hæmatemesis and the bloody stools of ulcer. Ulcer of the œsophagus is extremely rare in comparison to cancer.

From diverticulum, the cardia carcinoma is differentiated by the following facts.— Diverticulum is frequent in the upper third, rare in the lower third of the œsophagus. The permeability of the gullet will more variable than in cancer, because the sound will often skip the diverticulum. In the latter there will rarely be pain and the marasmus will not be so progressive and rapid. From Cardiospasmus or cramp of the cardia, the carcinoma is differentiated by the occasional free passage of the thickest tubes in the former, which occurs only in neurasthenics. Nutrition is not so much damaged as in cancer.

If tuberculosis or syphilis is present, one must think of the possibility of the neoplasm being caused by these diseases.

TREATMENT OF CARDIA CARCINOMA:—

As long as there is no cure possible, this must be palliative. During the time that deglutition still brings liquid food into to stomach, the sufferer must be carefully fed on highly nutritious liquid diet. Liquid eggs and wine as described in the diet of gastritis, beef tea, soups of fluid potatoe or pea purée in bouillon, Leube-Rosenthal beef solution, v. Mehrings Kraft chocolate, Egg nogg. When pain was great I have found that chloral hydrate gr. xv. t.i.d not only relieved it but acted as a local disinfectant in the diverticulum above the stenosis. Boas recommends iodide of potassium in doses of 15 grams 3 times daily as aiding in keeping the œsophagus from closing up as soon as it would otherwise. Arsenic is said to effect the same prolonged permeability.

In one case I succeeded in keeping the œsophagus open for six months by intubating with an inelastic tube 4 in. long and as wide as an ordinary Ewald tube. The tube was removed every 10 days and replaced. Patient lost no weight in those six months, but even gained. Death was caused by aspiration pneumonia during a period in which the tube was left out in order to rest the œsophagus from the stout cord with which the tube was connected with the mouth and was usually tied around patient's neck.

When deglutition is impossible the only thing left to be done is gastrostomy. If the patient can be persuaded to undergo this operation, it should be done before marasmus proceeds too far, as it then prolongs life much more and the shock of the operation is stood better.

This operation consists in making an opening into the stomach for purposes of feeding the patient by passing food directly into the organ. F. Kaiser (in *Czerny Beitr. z. operativ. chirurg.*) collected 31 gastrostomies, of these 28 died of the immediate results of the operation. Zesas (*Archiv f. Klin. Chirurg.* bd. 32, s. 188) reported 131 cases from literature, mostly œsophagus cancers which in their stenosing effects are identical with those of the cardia; among these only 19.5% recovered from the operation sufficiently to call this a success, because real cure is out of the question, it is a palliative measure only.

CARCINOMA OF THE BODY OF THE STOMACH.

CANCER OF THE FUNDUS, ANTERIOR OR POSTERIOR WALLS AND THE CURVATURES.

SUBJECTIVE SIGNS:—

1. Sudden abrupt beginning of the disease, striking an apparently healthy organ.
2. Loss of appetite in 90% of cases.
3. Aversion to meat.
4. In stenosing pyloric cancer there is much thirst.
5. Frequent eructations which, when there is dilatation can be very offensive.
6. Pressure in the beginning, pain later on.

7. There is frequently vomiting which is more copious in pyloric cancers because of the accumulations from the dilatation.

Frequently the vomit has a coffee ground appearance and the haemin test (referred to in part 1.) proves the presence of blood. The state of the bowels is variable. The vomit contains as a rule no hydrochloric acid, but excess of lactic acid.

OBJECTIVE SIGNS:—

On Inspection, Palpation and Percussion a tumor can be made out in at least 50% of the cases.

Tumors of the pylorus do not move with the respiratory movements unless attached to the liver; tumors of the curvatures generally show distinct respiratory movements.

EXAMINATION OF STOMACH CONTENTS:—

The results will be characteristic in most cases and evince.

1. Grave interference with the motility.

2. Suppression of secretion.

3. Products of stagnation dependent upon these. The disturbances of peristalsis are due most likely to a direct invasion of the muscularis by cancerous proliferation. The simplest way of testing the motor disturbance is to cleanse the stomach thoroughly by lavage in the evening, giving a test supper thereafter and examining the following morning when normally the stomach should be empty but in carcinoma much food and mucus with absence of hydrochloric acid and presence of lactic acid will in 88% of the cases be found. Hydrochloric acid is absent in 88% of the cases, in carcinomata that have arisen from old ulcers, there is claimed to be a secretion of hydrochloric acid until the last stages of the diseases. This assertion of Rosenheim's is not always correct as we have shown twice this winter at our clinic. If the glandular layer is invaded, secretion must cease no matter whether the carcinoma arose from an ulcer or not. Lactic acid is tested by Uffelmann's reaction, in carcinoma there is an excess in from 86 to 90%. Demonstration of the long, base ball bat shaped, Oppler—Boas bacilli is according to Kaufman, Schlesinger and Riegel, a very important sign. There should always be a careful lookout for histological evidences, bits

of the growth in the wash water and vomit, this clinches the diagnosis.

Secondary symptoms are anæmia and cachexia oedema of the ankles in 15 to 20% of the cases. The urine contains excess of nitrogen excretion, indican and pepton. Latent cancers may occur, they are very rarely observed however at the autopsy.

ULCUS CARCINOMATOSUM:—

The diagnosis is made from a history of ulcer, years of gastric pain, not a sudden abrupt beginning and presence of hydrochloric acid even hyperacidity — Hæmatemesis and blood in the stools in the previous history points to origin of the carcinoma from ulcer. Simple uncomplicated ulcer may cause a tumor-like thickening, simulating cancer; here the analysis of contents may even show excess of lactic acid owing to motor insufficiency and cicatricial stenosis and the diagnosis then becomes difficult. As is also the differential diagnosis of *ulcus carcinomatosum* from simple hypertrophic stenosis of pylorus, fortunately such states without any other important signs are rare.

TREATMENT:—

There is no successful medicinal treatment for this disease. Life may be most prolonged by a suitable diet, as nutritious as possible and adapted to the individual. A highly nutritious proteid carbohydrate and fatty diet should not be interdicted as long as the motility is good and the patient's strength can be upheld by intestinal digestion. Where there is stagnation owing to pyloric obstruction, the carbohydrates and fats must be diminished. The best tonic for the stomach is daily lavage, even where there is not much stagnation, but where the latter is marked and accompanied by fermentation, antiseptics must be added, such as boracic acid, 20 to 30 to 1000 H₂O, salicylic acid 3 to 1000 H₂O, sodium benzoate 10 to 30 to 1000 H₂O, Resorcin 10 to 30 to 1000 H₂O, Thymol 5 to 1000 H₂O, Lysol 1 to 2 to 1000 H₂O. Hydrochloric acid 4 to 5 to 1000 cc. H₂O. It is always well to get the stomach clean by using nothing but warm salt solution and finishing the lavage by the last irrigation with one of the disinfectants of which I prefer hydrochloric acid.

A tonic which has been serviceable in my experience and will

arouse appetite and promote digestion in the invaded organ, if this is at all possible, is the following;

R
Extract Condurango, fl. ʒxii.
Strychnin Sulphatis, gr. ⅓.
Hydrochloric Acid dil., gr. ⅓.
Elixir. Gentianæ, g. s. fl. ʒvi.
Sig. M.
Take ʒss. in ʒii aquæ after meals through a tube.

When there is much anæmia, the following formula has my preference in this disease as well as ulcer;

R
Solution of Iron and manganese, (*Parke, Davis & Co.*) fl. ʒvi.
Liquor Potassii arsenit min. XLVIII Misce. fl. ʒss. t.i.d.
Constipation is best met by large colon irrigations, Cascara Segrada, fl. Ext. or Syr. Activ. (*Clinton Pharmac. Co.*).
Diarrhœa must be met by Salol, Bismuth Salicylate, or Benz-naphthol. Opiates are not advisable for this symptom.

For pain, hot external cataplasms and 20 to 30 drops of comp. spir. of ether should be first tried. If severe, codein, gr. ¼ extract. Belladonnæ, gr. ⅓ in ʒi. peppermint water generally relieves it and may be repeated if requisite. The pain is rarely so intense as to require hypodermic injections of morphine. Lavage systematically and scientifically employed seems to prevent pain, it certainly prolongs life and sometimes apparently works wonders for these patients.

DIET FOR GASTRIC CARCINOMA.

At 8 A.M.

100 gr. milk with tea (67.5 + 50 gr.)	= 174. cal.
toast + 10 gr. butter	= 336.6. cal.

At 10 A.M.

100 gr. fried or broiled perch, pike or trout	= 72. Calories.
50 gr. toast	= 129. “

Instead of this calfs brain, sweet bread or 2 eggs are recommended for change.

At 12 M.

150 gr. milk rice	= 260. Calories.
100 gr. breast meat of fowl	= 142.45 “
50 gr. macaroni	= 126 “
200 gr. finest claret	= 100 “

When motility is good, mashed potatoes and purée of peas or beans are permissible.

At 3 P.M.

100 gr. tea with milk + 50 gr. cakes	= 254 Calories.
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At 7 P.M.

100 gr. cream	= 214.6 Calories.
50 gr. toast + 10 gr. butter + 30 gr. scraped raw beef or ham	= 376.3 “

At 9 P.M.

50 gr. rich milk or a glass of tokay, a few crackers, chocolate is permissible, young pigeon, partridge and prairie chicken also. If the motility is good one must not be too severe on the patient's desires for food, many cases can live and gain strength on a ordinary nourishing diet when it is not retained too long in the stomach; under these circumstances, mutton chops and beefsteak broiled after they are finely minced may be allowed.

SURGICAL TREATMENT:—

As already mentioned, gastrostomy is a palliative operation for malignant tumor of the cardia and œsophagus, to permit of direct introduction of food by establishing an opening between the stomach and the abdominal wall. In carcinoma of the pylorus, another palliative operation is practiced, where it is impossible or inexpedient to remove the growth, under the name of gastroenterostomy. This consists in the establishment of a new communication between the stomach and the small intestines, thus allowing the chyme to reach the intestines without passing the pylorus.

The *radical* operations are *resections* of the pylorus and excision of the tumor no matter where it may be situated in the stomach. These operations are contraindicated if metastases are detectable in other organs, by the presence of great anæmia or cachexia, large size of the tumor—adhesions to other organs. The detailed descrip-

tions of these operations belongs to text books on abdominal surgery;—

See surgery of the alimentary canal, by *A. Ernest Maylard*, P. Blakiston, Son and Co., Phila., 1896.

Abdominal surgery by *J. Geieg Smith*, published by P. Blakiston, Son and Co., Phila., 1896.

Surgery by American authors, by *Roswell Park*, vol. ii, chap. 8, published by Lea Bros., Phila.

System of surgery, by *Fred S. Dennis*, vol. iv, p. 217.

Abdominal surgery, by *M. H. Richardson* and *Farrar Cobb*.

A Text book of abdominal surgery, by *Skene* and *G. S. Keith*.

Fred'k Treve's Manual of operat. surgery, vol. ii, p. 405.

Franz Kænig, Lehrbuch d. speziel. chirurg., bd. ii, s. 281.

Penzoldt and *Stintzing's* Handbuch d. speziel. therapie, vol. iv, p. 444. The operative treatment of gastric disorders, by *Prof. von Heinecke*. Erlangen.

The following table gives the age in 2038 cases of gastric cancer, obtained from trustworthy sources and arranged according to decades: (*By William H. Welch, l.c.*).

Age.	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	90-100	over 100
No. of Cases.	2	55	271	499	620	428	140	20	2	1
Percent.	0.1	2.7	13.3	24.5	30.4	21	6.85	1	0.1	0.05

From this analysis we may conclude that three fourths of all gastric cancers occur between forty and seventy years. The absolutely largest number is found between fifty and sixty years, but, taking into consideration the number of those living, the liability to gastric cancer is as great between sixty and seventy years. Nevertheless the number of cases between thirty and forty years is considerable, and the occurrence of gastric cancer even between twenty and thirty is not so exceptional as is often represented, and is by no means to be ignored. The liability to gastric cancer seems to lessen after seventy years of age, but here the number of cases and the number of those living are so small that it is hazardous to draw positive conclusions.

MORBID ANATOMY:—

The following table gives the situation of the tumor in 1300 cases of cancer of the stomach: (*From article by Wm. H. Welch, l.c.*).

Pyloric Region.	Lesser curvature	Cardia.	Posterior Wall.	Whole or greater part of Stomach.	Multiple Tumors	Greater curvature	Anterior Wall.	Fundus.
791 60.8%	148 11.4%	104 8%	68 5.2%	61 4.7%	45 3.5%	34 2.6%	30 2.3%	19 1.5%

From this table it appears that three fifths of all gastric cancers occupy the pyloric region, but it is not to be understood that in all of these cases the pylorus itself is involved. In four fifths of the cases the comparatively small segment of the stomach represented by the cardia, the lesser curvature, and the pyloric region is the part affected by gastric cancer. The lesser curvature and the anterior and the posterior walls are involved more frequently than appears from the table, in as much as many cancers assigned to the pyloric region extends to these parts. The fundus is the least frequent seat of cancer. In the cases classified as involving the greater part of the stomach the fundus often escapes.

GASTRECTASIA.

MOTOR INSUFFICIENCY.

DILATATION OF THE STOMACH.

BOAS RECOGNIZES a *mechanical insufficiency of the first degree* (Mechanische Insufficienz ersten Grades) which is a myasthenia or atony of the gastric muscularis in which the ingesta remains in the stomach too long but finally are completely moved out into the intestines. There is no absolute retention of food but simply a delay in the expulsion. The fully developed dilatation Boas calls *mechanical insufficiency of the second degree*.

Riegel differentiates: — 1. *Simple Atony or Insufficiency of the stomach.*

2. *Atonic or typical ectasia or dilatation.*

3. *Secondary ectasia or pyloric stenosis with ectasia.*

Naunyn speaks simply of motor insufficiency and Rosenbach of mechanical stomach insufficiency. Schreiber, (Boas — *Archiv f. Verdauungs krankheiten*, bd. ii, p. 423) in attempting to select a designation which shall signify the most constantly present condition of all these morbid states of motility and one which shall unite them all around itself, reached and suggested the terms *Stasis stomach* (Stauungs-magen) with permanent digestion or permanently digesting stomach. Besides being a cumbersome circumlocution, the term does not even include all conditions of this type, for in Boas' mechanical insufficiency of the first degree and in Riegel's simple

Atony— conditions which I am convinced really do exist, there is certainly no permanent digestion.

Permanent digestion goes on in fully developed dilatations with impaired peristalsis as long as hydrochloric acid and ferments are secreted. But as there undoubtedly are long standing dilatations with complete achylia gastrica or loss of secretion, (Einhorn) there can be no digestion in them. The fact that the food is over-retained in them does not imply that it is digested, only in dilatations that show hydrochloric acid and ferments, can we speak of permanent digestion. The efforts of Schreiber to establish Reichmann's chronic secretion as a complication of dilatation with retained food products and permanent secretion caused by stimulation of the retained food are very convincing—we shall speak of the pathogenesis of gastrosuccorrhœa or Reichmann's disease under the nervous affections of the stomach. It is impossible however to invent a term which shall comprise the important features of all types of motor and mechanical insufficiency and probably as clear a classification as any is one based on Riegel and Boas.

1. Simple gastric atony or motor insufficiency or myasthenia without dilatation.

2. Atonic dilatation—motor insufficiency due to relaxation of the gastric walls, without pyloric stenosis.

3. Secondary dilatation due to pyloric stenosis.

It is self evident that these conditions may have widely different causes, but the one common sign is not the retention of food nor permanent digestion, but the impaired motility.

ETIOLOGY:—

Two kinds of cases may occur: either the atony of the gastric wall is not due to a mechanical obstacle; in this case nothing will oppose the free course of the contents, and they will only linger in the stomach because the latter is really incapable of ejecting them from its cavity in proper time, or the atony will be due to a pyloric stenosis; the muscular tonicity will have been overcome by an impassable obstacle, the fibres exhaust themselves in contending with a too strong resistance, and the dilatation may then be considered as following on existence of the obstacle. In the first case, the etiology is variable, and arises finally from a difficulty in the nutrition of the wall; in the other case it is purely mechanical.

DILATATION THROUGH A MECHANICAL OBSTACLE:—

The causes capable of opposing the passage of stomach contents into the intestine, and of opposing such a resistance to the contractions of the stomach that it dilates, are first of all the constrictions of the pylorus. These are generally the result of an anatomical alteration; cancer or cicatrices of circular ulcer, in the majority of cases, determine the constriction of the pyloric ring.

To these causes must be added a proliferation of the muscular fibres and of the tissue adjoining this ring, a veritable tumor, benign in character, but the mechanical effects of which are practically analogous to those of the ulcer and of the cancer. Nauwerk was one of the first to draw attention to these facts. A spasm of the pylorus, which can be compared to a spasm of the sphincter of the anus, can just as well determine an occlusion of this orifice. This spasm, which has been admitted by authors, for a long time, for reasons a little theoretical perhaps, has been demonstrated since gastric surgery has permitted the more direct exploration of this organ. Martin, among others, has seen a pylorus, large enough to admit the passage of two fingers, bring on a considerable dilatation by its spasmodic occlusion consequent upon a circular ulcer after which had occurred a considerable hyperacidity. Lastly, just as there is said to exist a pure mitral constriction of congenital origin, so Landour is said to have proved the existence of a pure pyloric constriction. He is said to have collected ten observations, and to have shown that this orifice, large enough during infancy, might undergo an arrest of development and remain very small, while the stomach grows larger with age, a serious dilatation would result from this conjunction of circumstances.

However, the obstacle need not necessarily have its seat in the tissue of the pylorus itself. We have elsewhere said that a polypus inserted in the gastric cavity was capable of bringing about a dilatation by becoming fixed more or less in the intestinal orifice, and thus causing its occlusion. Deiters has collected at the anatomical-pathological institute of Greifswald a somewhat large number of observations in which congenital malformations, abnormal foldings, diverticulae, and atresia had provoked dilatations by constricting the intestine in the more or less immediate vicinity of the pylorus. An anatomical lesion of the duodenum, the cicatrix of an ulcer, for

example, would produce the same effects by diminishing the calibre of the passage.

There exists also a whole series of obstacles of extrinsic origin, which by compression of the pylorus or of the under-lying portion of the intestine, have a tendency to bring about the same consequences as the causes hitherto mentioned.

There are, firstly; peritoneal adhesions, circumscribed or not, the results of former inflammations. The cicatrix of a circular ulcer may be placed in such a way that it may not be able to hinder directly the course of the chyme over its own location, but the fibrous tracts which radiate from its basis as a rule contract more every day, and end by obliterating the lumen either directly, or by making the under-lying intestine describe an abnormal curve.

The liver and the pancreas are equally, (in some cases) the starting-point of inflammations which will leave in their train, similar anatomical modifications. The head of the pancreas, so intimately connected with the duodenum, may become cystic or cancerous, and cause by compression a duodenal stenosis with following dilatation of the stomach. A congenital displacement of the same organ would bring about the same disorders. (Cechini). A biliary concretion, by dilating the ampulla of Vater, or by compressing the intestinal wall, may produce a compression of the duodenum sufficient to bring about gastric dilatation; Grundzach has recently reported a case of this kind. Bartels, Mueller, Warnek, Litten and other authors have studied the relations of ectopy of the right kidney and of gastric dilatation. These studies have been continued in an interesting work by Bruhl, and Mathieu has also just recently reported new cases (*Societe Medical d' Hospitaux*). The persons presenting this coincidence of movable kidney and dilatation of the stomach, are usually young girls or women of the working class, who are in the habit of fixing their skirts at the level of their hips with laces very tightly drawn, causing an external constriction, which is shown by the presence of a permanent furrow. In other cases, men, who wear a belt or strap, produce the same results. The right kidney thus becomes displaced forwards and inwards, pressing upon the fixed, descending portion of the duodenum, which is situated between the hilum of the kidney and the vertebral column. Such partial obliteration of the intestine would bring about a slower

and more difficult evacuation of the contents of the stomach. As food would remain longer in the stomach, there would result first an increase of activity and a slight hypertrophy, then later, a muscular relaxation, a distention of the walls and a dilatation of the cavity of the organ.

Ewald and Pertick have gathered together a certain number of cases in which a hernia of the floating portion of the duodenum, or of the first part of the jejunum across a laceration of the mesentery, or a diverticulum of these parts of the intestine, has played the same part, and caused in the end a gastric stasis.

DILATATION WITHOUT MECHANICAL CAUSES:—

Besides the cases in which mechanical obstruction causes a dilatation, one sees gastric atony follow upon a whole series of general affections. Weakness of the walls is in these the only cause which can be supposed, and these cases constitute the second group of which we spoke at the beginning.

Dilatations of this class which seem to be primary, may be acute or chronic. The first kind have for their cause either a traumatism, or a surgical intervention (laparotomy,) or else a serious infectious disease; Bartels, Hilton Fagge, Lepoil and Montaya have cited examples in which typhoid fever seems to have played the part of the chief cause. In this case generally, the dilatation seems due to the loss of tonicity of the musculature of the stomach and of the abdomen. In other cases, the origin of the evil is an excess of food, an error committed so frequently by convalescents confined to one diet for a long time.

Chronic dilatations are dependent upon a great number of factors. First of all, large eaters suffer first with distention of the stomach, then later with dilatation; this phenomenon is comparatively frequent with persons into whose ordinary diet enters a large quantity of liquids: such is the case with excessive drinkers of beer, which acts not only mechanically by its volume, but also through the irritating and poisonous substances with which it may be adulterated. The milk treatment might just as well, when ill administered, bring about a distension, and later a dilatation of the stomach by a superabundance of liquid food; Debove has especially drawn attention to the drawbacks, of prescribing milk in considerable quantities, and

has cited, among others, a case of circular ulcer cured by the daily allowance of eight litres of milk; but an enormous dilatation of the stomach resulted. In a general way, it must be admitted that over-feeding produces a certain amount of inflammation. The dilatation is produced under this double influence of the catarrh and of the distention; without the addition of the first of these causes, megalogastria alone would occur.

We have seen that chronic catarrh or simple chronic gastritis can have as its consequence a considerable atrophy of the muscular fibres of the stomach, and that an enormous dilatation of the stomach is sometimes the result. The same may be said of dyspepsia, provided that it is one of the forms where a hyperacid secretion causes a prolonged stasis of the amylaceous substances; such cases have been collected by Mathieu and Remond, under the name of dyspepsia with organic hyperacidity and stasis. In other cases muscular atony is the result of a prolonged stay in the stomach of undigested food, with fermentation thereof, when hydrochloric acid is wanting. Drawn out by a weight more or less considerable, and distended by the gases that are developed in the putrefying mass, the muscular fibres lose the faculty of withdrawing back upon themselves. Furthermore, since the glands become diseased, and since the inflammation is not limited, as we have shown, to the glandular tunic alone, but on the contrary invades to a variable degree, it is true the body of the wall, one must not be surprised to see the atony very soon become difficult to cure. The dilatation found in consumptives, in chlorosis, etc., is caused solely by chronic gastritis, which is caused by blood alterations, the results of these diseases. In diabetes, both the chronic gastritis and superabundance of food, take part in the alteration of the walls and may finally lead to amyloid and colloid degenerations of the muscular fibres.

Atony which is purely nervous, concerning which Germain, see and Mathieu have published numerous researches, is produced in consequence of crises appearing to indicate the successive and alternating intervention of a particular state of spasm, and of atony of the gastro-intestinal system. These crises are produced by an occasional and general cause such as sad emotions, mental shock, etc. Neurasthenia then takes part in the etiology of dilatation, in the same way in which we have seen it become one of the import-

ant factors in syndrome dyspepsia. The atony due to neurasthenia can be just as well brought about by lesion of the central or peripheral nervous system, and the dilatation will then depend on a deep seated alteration either of the centres, or of the peripheral nerves. Glenard has represented general ptosis of the abdominal organs, as the expression of a particular diathesis, a condition of relaxation of the tissues with smooth muscular fibres, and has considered the dilatation as depending upon this general state. Although we have not verified the lesions upon which Glenard has built this theory, we cannot deny the existence in clinical medicine of cases analogous to those described by this author; such are the dilatations through nephroptosis.

PATHOLOGICAL ANATOMY:—

If the etiology of dilatation is complex, its pathological anatomy is simple, and to a slightly variable degree, the lesion is always the same. At the autopsy of a subject dead from cancer of the pylorus, for instance, one finds the abdomen filled by a voluminous sac which comes down more or less near the pubes. This sac, which represents the stomach having lost all its normal relations, and excessively dilated, may contain enormous quantities of liquid, and the ancient authors who knew only the extreme cases, have cited extraordinary examples of this. Plempius is said to have seen a stomach that held nine pints of liquid; Stengel mentions a stomach containing twelve "measures"; Schurig, a stomach containing forty-eight litres; Henricus ab Herr found a stomach which filled the whole of the abdomen.

The walls of this gastric sac have become thin; in general, this thinness is found in all the coats, and with the microscope one finds an atrophy of the mucosa; at the same time, the submucosa is now composed only of isolated bunches of muscular fibres separated by the connective tissue. When the dilatation is caused by an obstacle at the pylorus, hypertrophy of the muscular wall is produced first; then a veritable interstitial sclerosis comes on little by little submerging the true elements, and the atony of the wall is due to the disappearance of the contractile fibres; an apparent hypertrophy through exaggerated proliferation of the connective tissue, sometimes masks in these cases the actual atrophy. The muscular hy-

peritrophy continues very long in the pyloric region where it also attains its maximum point, and we have seen, while studying the ulcer, the increase of resistance and of the volume of the walls, which sometimes results, may simulate a tumor.

A stomach which has undergone acute dilatation may present veritable linear thickenings of the serous coat. This modification precedes the rupture of the organ, the symptoms of which we shall have occasion to describe further on.

The anatomical-pathological study of a dilated stomach includes naturally that of the causes of the dilatation which we have already described. A dilated stomach may present variable forms due to the action of a special cause. If a cicatricial or scirrhus constriction causes the cardia and the pylorus to approach each other, the stomach will be pyriform; but if the same lesion has ploughed a furrow, more or less deep, on the wall, a dilatation in the shape of an hour-glass will be produced. But these are anatomical curiosities, and the symptoms do not differ from those caused by occlusion of the pylorus.

SYMPTOMS.

STATE OF THE APPETITE:—

The symptoms of dilatation are very variable. The appetite may have completely disappeared, or have become considerably decreased, some patients, for instance, will not need more than one meal a day. In other cases, since the stomach merely plays the part of a reservoir with no outlet, and the foods being no longer evacuated from the stomach into the intestine, digestion and absorption occur no longer; the patients may be tormented with hunger, and they are in an analogous situation, as far as effects are concerned, to that of persons seized with an impassable stenosis of the œsophagus. They try then to satisfy their appetite, and yielding to the solicitation of hunger, present veritable bulimic phenomena. In reality, it is not hard to understand this difference which depends, practically, on the nature of the obstacle to the course of the foods; anorexia is observed chiefly in cancerous patients and those seized with chronic gastritis; while a cicatrix of a circular ulcer may have obliterated the pylorus without bringing about very clear modifications of the special-sensibility of the stomach.

PAIN:—

Pain is not usually very acute; it consists rather of a sensation of fullness and of tension, which, as far as the pain is concerned, is not to be compared to acute pain observed in ulcers and cancer. Sometimes, as in the form described by See and Mathieu, painful crises precede the moment when the dilatation becomes appreciable; the same may be said of the dilatation following on Reichmann's disease. Lastly, an ulcer or a cancer can add their peculiar pains to the somewhat painful sensation which exists when the stomach is not emptied, for any reason.

GASEOUS DISCHARGES:—

When the dilatation is not very marked, especially in neurasthenic patients, the epigastric heaviness and the swelling give way little by little, and if the patient takes his meals at regular intervals, his stomach at last empties itself, and his pains disappear. But generally they cease only when vomitings more or less copious, have relieved the gastric cavity of the foods which have burdened it, sometimes for more than twenty-four hours.

To this feeling of fullness are added disgusting gaseous discharges, often very fetid. The alimentary contents in fact are liable to set free in considerable quantity many different gases. We have cited some examples of these cases in the chapter on general symptomatology above, and Franz Kuhn has published a long work on this subject. The principal gases are carbonic acid, hydrogen, oxygen, nitrogen, carburet of hydrogen and carbonic dioxide. Whenever there is stasis, their presence may be verified by directly extracting the gas from the stomach by appropriate means, or by setting free the gastric contents in a closed vessel, after having extracted them artificially. The causes of this gaseous development are supposed to be the presence of fungi resisting the antiseptic action of the hydrochloric acid even when present in excess, the fungi have been isolated and cultivated; they may be associated with a great number of other kinds of fungi when the contents of the stomach become neutral or alkaline. These fermentations, which are very frequent, are moderated by Salicylic acid or saccharine; boric acid, carbolic acid; — creosote and chlorine water have no effect except in doses which are incompatible with their therapeutic

use. The great quantity of liquid contained in the stomach facilitates the development of anaerobic germs, giving rise to products of fermentation, more complex and perhaps more poisonous.

PYROSIS:—

The regurgitation of a certain quantity of liquid often accompanies the gas discharges which pass through the cardia, and pyrosis which, as we have seen, results from contact of the majority of the neutral, acid or alkaline gastric liquids, with the wall of the œsophagus, is often added to the other phenomena which torment the patient.

VOMITING:—

However, the gas discharges and the pyrosis are relieved, just as the sensation of fulness is, by the emesis which relieves the stomach of its contents. They are not as frequent as they are at certain stages of the development of cancer or of ulcer; they are generally separated from each other by a variable but comparatively long time, and it is rarely that they occur at the time of the maximum of digestion. The following is the course which matters usually take:—for one or two days a patient has suffered, after each meal, from a sensation of growing uneasiness, and from a feeling of weight at the epigastrium, more and more painful; then suddenly, often towards the middle of the night, he is seized by very abundant vomitings, after which he can enjoy a little rest.

These vomitings are sometimes composed of several litres of a mixture of solid food, drinks and mucus. The quantity of vomited matter is a first rate symptom of dilatation, and allows it to be distinguished, for instance, from simple displacements of the stomach. We have already said that chronic gastritis, cancer, etc., might give way to slight hemorrhages, and in this case, the blood very much modified, remains a long while in the stomach: the same phenomenon will be recognized in dilatation.

The permanent presence of bile and of pancreatic fluid, of which we have given the characteristics, would allow us to decide on stenosis of the duodenum, and would be an excellent symptom of dilations following on the compression of this part of the intestine by a movable kidney, for instance. Lastly, the vomited matter will have a more offensive smell, the longer it has staid in the stomach.

Later on, when the walls are distended, the vomitings come on at greater intervals; the odor of substances vomited becomes more revolting; and now the emesis is rarely sufficient to empty the stomach, the feeling of relief, which at first followed, ceases to be produced.

GENERAL STATE OF HEALTH:—

The general state of health suffers naturally from digestive disturbances, but it is more often, and more deeply influenced by the cause of the dilatation than by the dilatation itself. Diabetes, chlorosis and great pyrexias provoke general disorders, and it is difficult to distinguish from among these disturbances, that which belongs properly to gastric atony. Neurasthenia often causes an atony of the muscular fibres of the stomach, the consequences of which cannot but have a marked influence on the nutrition of the patient and encourage and develop in their turn an original neurasthenia. There is thus produced a veritable pernicious circle in which, however, the general cause seems to play the most prominent part, since, as we have said with regard to dyspepsia, it is only by addressing one-self to it, that one has any chance of curing the patient.

Bouchard and his pupils have seen in this simple or original dilatation the pathogenic cause of a great number of very different diseases. We have observed nothing which allows us to accept their view. We therefore cannot appreciate the value of the theories which they have built up, and which are to be found remarkably set forth in the works on general pathology by Bouchard, and the thesis of Le Gendre. Personally we hold to the idea that pathological phenomena of this kind, of which the stomach may be the seat, are most often, if not always secondary.

In dilatation through an organic cause, the disturbance of the general state will vary with this cause. It is thus observed that in cancerous patients the dilatation is accompanied by the most evident cachexia. In the ulcer, Reichmann's disease and chronic gastritis, it will only be coincident with an emaciation more or less marked.

In the case of children, Moncorro and Cornby have attributed to dilatation caused by overfeeding a certain part of the develop-

ment of rachitis. The latter author also considers it to be the cause of certain convulsions, of insomnia, of ring-worms, of urticaria and of bronchitis. We have never observed anything of this kind.

The state of the tongue is not necessarily in agreement with that of the gastric cavity, and if it is usually coated and white, this symptom, like many others, depends much more upon local states and on the general state of health than on the gastrectasia itself. We have spoken fully of the various state of the tongue in the chapter on chronic gastritis.

CARDIO—PULMONARY SYMPTOMS:—

The distention of the gastric cavity by gases in food sometimes hinders considerably the functions of the diaphragm, and disturbs the action of the respiratory apparatus and of the circulation, more or less in different cases; one meets most often with dyspnœic phenomena, or modifications in the sound of the heart and in the rhythm of the pulse.

Mattheides has gathered together a certain number of cases in which was to be observed a sensation analogous to that of globus hystericus in patients afflicted by dilatation. He has shown that this sensation was aggravated when the stomach had sunken, and diminished, on the other hand, when it had risen; he had concluded from this that the displacement of the stomach so often accompanying the dilatation of this organ was the cause of this sensation of globus through the dragging which occurs on the œsophagus. Schmidt is said to have verified, by a laparotomy, the existence of these anatomical disorders in a patient who had previously complained of the sensation of globus, but our personal observations lead us to believe that there is no relation at all between dilatation of the stomach and globus hystericus.

PERCUSSION AND PALPATION OF THE STOMACH:—

Percussion and palpation allow us to ascertain the limits of the lower edge of the great curvature, and thus to appreciate the degree of the ectasia. The percussion should be performed with the patient standing up and again lying on his back. The measured ingestion of a certain quantity of water will allow one to estimate the atony of the wall, and at the same time will furnish exact data on the dis-

placement of the lower edge of the organ, which has become heavy. However, since difficulties depending on the more or less noticeable repletion of the intestine might hinder the interpretation of results, it is best to distend the stomach still further with an effervescing mixture, at the same time filling the colon by an injection. Other authors have proposed to perform the operation inversely, and percuss the stomach made heavy by a certain quantity of water, while the colon is distended by gas. (Ewald). It is true that these excessive precautions would make mistakes very difficult. Professor Osler holds, that when the distended stomach is outlined on the wall, one can usually follow its delineations with the eye, and of course much better by percussion. In the *Phila. Med. Times* for May, 1891—Pepper reports a case of dilatation caused by Schirrous of the pylorus in which there was a visible peristalsis.

This gaseous distension has also the advantage that it allows the distinction to be made between true dilatation and a simple displacement of the organ.

By palpation the splashing sound can be investigated. This is easy to perceive when the stomach, the pylorus of which is constricted, is full of those liquid masses already mentioned in connection with the vomitings. But when the dilatation is not very marked, the splashing becomes less clear, and Debove has recently shown that the intestinal canals when half distended by gases are capable, under the influence of movements communicated by the fingers, of producing a sound so like that of the splashing, as to make the distinction very difficult. Chomele had already drawn attention to this cause of mistakes, and to that which depends on the presence of liquid and gas in the large intestine: "The splashing in the stomach" says he, "might be confused with a similar sound of which the large intestine is sometimes the seat, which can be produced by the lateral movement of the body, but still more easily by the pressure of the hand on the regions occupied by the colon". It is met with especially in subjects who have recently received an injection, and those who have been seized with serous diarrhoea. The knowledge of these conditions, and the particular source of the splashing sound are sufficient to distinguish it from intestinal splashing. In fact many authors have met with a splashing sound even when, on introducing the probe into the stomach, they have been unable to

withdraw any liquid whatever. Jaworski has recently brought to notice four cases of this kind, and we have observed many examples of it. It is not, therefore, a reliable sign, and one must guard against attaching more importance to it than it really deserves.

One can also perceive the lower limit of the stomach, either by means of the sound, the end of which one seeks to feel through the abdominal wall, after having slowly advanced it as far as possible; or by means of special instruments such as that invented by Ghibaut of Nancy, which consists of a probe through which slides a thread with a leaden weight. The probe is long enough to reach the cardia, and the quantity of thread taken by the leaden weight before it arrives at the bottom of the stomach, allows one to measure the vertical dimension of the gastric cavity.

We have described the methods of procedure based on the employment of salol, oil, iodide of potassium, etc., destined to determine the state of the motor functions and of the absorption of the mucous membrane. In dilatation, they give information of varying value, but inferior to that furnished by exploration with the sound. We have already said by what criterion we recognize atony of a muscular wall, either presence of *debris* of food in the morning before breakfast, or the prolonged stay, in the gastric cavity, of a trial meal. The Hemmeter Gastrograph is a graphic method of obtaining motor records from the human stomach, and its results are generally reliable. (Part 1. p. 54).

TEST MEALS:—

In order to make a patient absorb a test meal, the study of which may procure some information, care must be taken to wash the gastric cavity, on the evening of the day before. The substances extracted by this preliminary injection are sometimes very abundant and having the same composition as those vomited, generally become separated, when allowed to stand, into three layers: an upper one frothy and turbid, a middle one liquid, and a lower one composed of alimentary detritus of all kinds, or simply of amylaceous substances, as occurs in the case of patients suffering from hyperacidity. With the microscope, ferments and sarcinæ will be discovered, and all the series of products which can, normally or abnormally, be contained in the stomach.

In the morning before breakfast, the gastric cavity, which has been previously cleansed the evening before, may again contain products of secretion, which are acid, (gastrosuccorrhea) neutral or rich in mucus, (chronic gastritis, and alcoholism). The digestion of the test meal will be generally slow, and especially in cases of cancer, it will be impossible to detect free hydrochloric acid. In other patients a normal or exaggerated state of secretion of a hydrochloric acid will be found. In all cases the appropriate re-agents allow us to recognize the presence in excess, of lactic acid, and fatty acid products.

CONSTIPATION:—

To these already numerous symptoms are to be added those which come from the state of the intestines. Constipation is frequent and obstinate, and not only are the stools rare, but the quantity of substances evacuated is also much less than in the normal state. This is a very valuable indication, for it shows the approximate amount of food which passes into the intestine. Thus Kussmaul had already been able to establish the prognosis of the patients attended by him, according as, in treating the dilatations, the normal course of the food substances was re-established or not. The latter state indicates an incurable stenosis of the pylorus.

STATE OF THE URINE:—

The urine is equally modified in quantity and in quality. The patients are in a veritable state of chronic inanition, and the urea is therefore necessarily diminished. The stomach absorbs little liquid, as the constant thirst by which these patients are tormented testifies; and the dilatation brings about a deficient urinary secretion. Lastly, when the dilatation accompanies an excessive secretion of hydrochloric acid, and the latter is thrown out, either by frequent vomitings, or by injections too often repeated, the urine becomes alkaline. We have already sufficiently developed this special urology, in the first part of this work.

DIAGNOSIS:—

The diagnosis of dilatation of the stomach, results from the study of the various symptoms analyzed above, and it seems difficult for a mistake to occur if several of them are observed simul-

taneously. However, no sign, unless it is the presence in notable quantity of food in the stomach before breakfast, is pathognomic; still when one meets with the other symptoms singly, one is much more liable to admit a dilatation which does not really exist, than to misunderstand one which has remained latent. In order to make the diagnosis more easy, recourse has been had too strange and sometimes dangerous methods of procedure. It is thus that Bugge had the idea of recommending the following operation; he determines by percussion, the patient standing up, the lower edge of the stomach, and drives the needle of a pravaz syringe above the discovered limit. If the liquid extracted is acid, the stomach has really been reached. Rosenbach, in order to measure the energy of a gastric musculature, when the dilatation is as yet hardly outlined, seeks to fix the level of the liquid contained or poured into the organ. Starting with the idea that the ascent or descent of the surface of the liquid furnishes the best information on the contractile power of the stomach. When the quantity of water deposited overcomes this force, the level is lowered in spite of the quantity of this water; in the contrary case it rises, so that it can be seen in any cavity whether it is formed of extensible walls or not. To ascertain the limits of the level, Rosenbach auscultates the stomach in the following way; by means of a rubber ball, he makes a few bubbles of air enter the stomach, by force, through the tube. When the lower orifice plunges into the liquid, a loud bubbling sound is produced, sometimes metallic. If the orifice remains above the liquid, nothing is heard but the whistling of the air expelled. It is enough then to withdraw or advance the probe, to know whether the level rises or falls when water is added and at the same time, to measure the lowering of the great curvature. In an individual whose stomach is dilated, 500 cubic centimeters of liquid, cause only an insensible elevation of the level; 1000 cubic centimeters sometimes cause it to fall; on the contrary in healthy individuals, this phenomenon only occurs if ingestion of the liquid masses is sudden. Sigmund Purjerz has proposed to adapt a manometer to the tube; while the extremity of the latter is in the œsophagus, one verifies negative variations of pressure. It becomes positive when the cardia is passed; this moment is noted, and also the one when the probe cannot be pushed any further. The length of tube that was necessary to use

measures the vertical dimension of the gastric cavity.

It must be agreed that these methods, although interesting as physical experiments, are not very practical, and we prefer the simple exploration by means of the sound, associated or not, with the artificial gaseous distension of the stomach. These methods, in fact, suffice, and more than suffice to distinguish a dilated stomach from a displaced stomach, and from a naturally large stomach. But if they do not the following methods of Einhorn and Hemmeter will leave no room for doubt.

GATROPTOSIS—DISPLACEMENT OF THE STOMACH:—

The pylorus may be displaced, and freely movable below the epigastric region, without in reality causing any gastric disturbance. One perceives, by palpation and percussion, the great curvature of the stomach below the umbilicus. One might then be very much disposed to affirm it to be dilated, while, by setting free carbonic acid in the gastric cavity, one sees not only the great, but also the small curvature outlined under the skin, and one can trace the outline of the whole stomach, on the abdominal wall, following the limits of the corresponding sonorous zone. This dislocation of the pylorus, frequent in neurasthenic patients, which Ewald has emphasized, and which we ourselves have had occasion to observe several times, is one of the reasons which causes dilatation to be so easily diagnosed in these patients, who think their stomach is suffering when it is not diseased. One cannot be certain of this displacement without making use of effervescing mixtures and the electro-diaphane or the Hemmeter intragastric bag; by these it is to be distinguished from cases of megalogastria. In both cases the stomach is empty in the morning before breakfast; but in those where there is a lowering of the pylorus, one finds, on using the different means of exploration, a lowering of the great curvature, whilst the small one has kept its normal relations. In both circumstances evacuation of the stomach, in the lapse of a normal time, indicates that the tonicity of the muscle is preserved.

The differential diagnosis between gastropptosis or prolapsus of the stomach, gastrectasia or dilatation and megalogastria or large stomach (giant stomach) can be facilitated by distending the organ with air or gas, but even here the success, inspection, palpation

and percussion will depend upon the thickness and resistance of the external abdominal wall. Where there is very little or no emaciation, it is by no means easy to palpate through the abdominal wall. Then again much gas escapes into the intestine when the stomach is distended by effervescent mixtures.

But by means of the stomach-shaped intragastric rubber bag, which was described on page 54, or by Einhorn's electro-diaphane (page 78) it is possible to make the differential diagnosis without much difficulty. By Hemmeter's apparatus, which was originally designed to obtain records of the gastric peristalsis, it is also possible to measure the capacity of the stomach by determining the amount of air required to distend it within the stomach (see first part on this subject). It was first used for this purpose by Schreiber of Königsberg. This will at once enable one to diagnose a dilated stomach from one which has prolapsed but retained its normal capacity. Einhorn's diaphane is a practical method for demonstrating these two conditions to the eye. The Roentgen rays are also available for the same purpose as I have demonstrated (*Boston Medical and Surgical Journal*, 1896). The greater curvature may be outlined by photographing a metallic spiral electrode that has been introduced and made to apply along the greater curvature, according to suggestions first made by Wegele; or the stomach may be photographed into by my method, which consists in distending the stomach by my intragastric stomach-shaped bag, the inner surface of which has been previously coated by a deposit of plumbic acetate or nitrate of silver, which is poured into the bag in a saturated solution, and allowed to dry on the inside. The bag is then introduced and distended by air filling out exactly the entire stomach. The thin coating of plumbic acetate cuts off the Roentgen rays sufficiently to obtain a photograph. This method is troublesome, and can be satisfactorily executed only in hospital practice. For private practice the Einhorn electro-diaphane is most expedient, as it permits of a diagnosis to be made by inspection. Debove and Rémond (*Maladies de l'estomac*, p. 87) state that this method is difficult of execution, and imposes much suffering upon the patient. From what we have seen almost weekly with Einhorn's apparatus, we differ emphatically from these observers, and believe a further experience with the apparatus will effect a change in their opinion.

DIAGNOSIS OF THE CAUSE:—

When one is quite certain that it is a case of dilatation, there still remains to diagnose the cause of this dilatation.

The former history of the patient, the examination of the substances vomited, and the results furnished by a test meal, would then provide the principal data. The representations differ, in fact, considerably according as one finds a dilatation of cancerous origin, or one caused by ulcer or gastritis. A former poison and inveterate alcoholism will make one think of a cicatricial adhesion of the pylorus, of that particular atony of the submucosa, that one finds in chronic gastritis. Lastly, in studying the test meal, if there is hydrochloric acid in normal or excessive quantity one can eliminate cancer with almost certainty, especially if the substances vomited contain, at the same time, bile. If the bile is always wanting in the substances vomited, or in the gastric contents, either before or after eating, one will have a right to think of constriction of the pylorus; the latter will probably be of a cancerous origin, if the hydrochloric acid were wanting at the same time.

On the contrary, the constant presence of bile and of pancreatic juice in the stomach would be a proof that the dilatation is a consequence of an occlusion of the duodenum, below Vater's ampulla; this occlusion may result from a movable kidney, a fibrous adhesion, gall stones, pancreatic neoplasm, etc.

One can then discover, in this way, whether the pylorus is the seat of an obstacle, or whether it is passable, and lastly, what is the probable nature of this obstacle. The accuracy of these results, it is true, is by no means absolute, but in practice, in associating them with other data furnished by the elements of the diagnosis of each gastric affection, one arrives at a largely sufficient approximation. A stenosis of the duodenum can be definitely ascertained by my method of duodenal intubation (see part 1. p. 31).

PROGNOSIS:—

The evolution of the disease which occupies us, varies according to the cause; when it is a case of simple atony of recent date, a proper treatment of which we shall speak again further on, may bring amelioration rapidly, and even cure. But when it is a case of dilatation with atrophy of the muscular coat, especially when there

exists an impassable obstacle at the pylorus, the cure is impossible, except sometimes by operation. The treatment still relieves the painful phenomena but the inanition makes progress from day to day, and the patient succumbs to inanition, unless one of the complications that we have mentioned comes and hastens the end.

MALFORMATION OF THE GASTRIC CAVITY:—

Dilatation is, in reality, a deformity of the stomach. Under this head comes the study of a certain number of cases in which malformations or changes of form, of the most varied kind, have been verified.

Atresia of the gastric cavity, results from diminution of work by the organ, through insufficiency of alimentary contributions. Inanition and constriction of the œsophagus or of the cardia, will thus have been the first cause of this atrophy. In other cases it is a cancerous infiltration extended over the whole wall, a chronic gastritis with hypertrophy of the submucosa, and of the connective tissue (Linitis plastica), or a fibrous, deforming peritonitis, which will have played the same part. The calibre of the stomach thus narrowed sometimes does not exceed that of the intestine. When the upper digestive paths are open, vomitings occur which appear as soon as the quantity of food exceeds the very small volume that the stomach can contain, and the small calibre of this organ, becomes still more clear when one comes to distending it with carbonic acid. If stenosis of the œsophagus, or of the cardia, exists, the passage of the probe becomes impossible, and the symptoms of these constrictions assume enough importance to obscure entirely those which might be furnished by the state of the stomach.

THE HOUR-GLASS STOMACH:—

Cicatrization from an ulcer, or from the loss of any substance, may bring about malformations which will present, in some cases, an analogous appearance to certain congenital malformations. Stoker has published one of these cases, where the stomach, divided into two parts by a congenital furrow, had never during life, presented any functional disturbance. Iago has related the story of a patient who succumbed when 42 years old, after having presented for ten months, uncontrollable vomitings; on the examination of

the abdomen, there was found, underneath the liver, a soft tumor which had been taken for the right displaced kidney. There existed no tumor at the level of the pylorus; the vomitings took place without pain, and were not preceded by regurgitation, there was no cachexia to be found. At the autopsy there was found a stomach presenting two dilated sacks which communicated by a closed narrow passage situated about the middle of the organ; the index finger could not pass this constriction; the cicatrices which had produced this deformity, must have been caused by a former disease which appeared at the age of 30, and was characterized by hematemesis and acute pains. A patient of 50 years old, observed by Luigi Mazotti, experienced such intense pains, after meals, that she used to squirm on her bed, and only found little relief after having vomited everything that she had just taken. At the autopsy, the stomach was found divided into two parts: the upper one vertical, the lower one, directed horizontally towards the right side; a narrow passage was situated between the two parts. The lower portion of the stomach, had made a complete circle, and the contracted point was exactly the centre around which this rotation had occurred. The upper part of the stomach was distended by gases; the lower one was empty and joined to the abdominal wall by some adhesions. When the viscus had been replaced in its normal position, it was found that neither the orifices nor the wall presented any modification, and it was impossible to discover the cause of this displacement. Chiari suspected a cancerous constriction of the pylorus, in a patient who, in reality, had an intussusception of the stomach into the duodenum.

These cases are rare, and could not be diagnosed. It is sufficient, therefore, for us to have mentioned them. Details could be found in the recent memoirs of Bauermeister and Saundby and in the thesis of Kern. (*Inaug. Dissect. Berlin*, 1881). Chiari (*Wien Med. Wochschr.*, No. 42, 1890). Bauermeister (*Inaug. Dissect. Halle*, 1790).

TREATMENT OF MOTOR INSUFFICIENCY OF THE FIRST
DEGREE — GASTRIC ATONY OR MYASTHENIA.

PROPHYLAXIS:—

The muscularis of the digestive organs may be weak by inheritance. Chlorosis, Anæmia, Tuberculosis and Cholelithiasis, exhausting hemorrhages, infectious diseases, typhoid, malaria diphtheria influenza may bring on myasthenia and frequent and rapidly consecutive births may by causing increase of space in the aedominal cavity and loss of tone to the abdominal muscles — lead up to gastric atony. Bad chewing, hasting eating and deglutition, defective teeth must have their correction. The treatment in all cases must seek and adapt itself to removal of the cause. Anaemia must be treated by proper food, peptonate of iron, bone marrow and arsenic. In ladies with gastropstosis and atony, the abdominal muscles must be strengthened and supported by proper bandages. The treatment proper includes diet, hydropathic, electric proceeedures, massage and medicines.

DIET:—

Patients with gastric atony must eat frequently but very little at a time. As water is not absorbed from the stomach the quantity of liquids must not exceed 1 – 1½ quarts in 24 hours, including all drinks, coffee, soups, etc. Where there is a craving for more liquid than this, it should be introduced by enema.

MILK CURES:—

My experience with the frequent and peristent administration of milk as observed in milk cure sanitariums in Germany is discouraging. I believe this treatment to be a useless dietetic experiment in gastric atony.

The special diet must be selected according to the state of the gastric secretions. If there is hyperacidity, I recommend a vigorous beef and mutton diet with limited carbohydrates, hard and soft boiled eggs, ham, tongue. oysters, duck, deer in every form; of vegetables, I allow carrots, spinach, soft boiled turnips, beans, peas, cauliflower. Potatce, macaroni, rice and farina gruel are permissable.

I am not too strict concerning alcoholics, and where a trial proves that they aid digestion, I generally permit claret, rhine wine, and even small amounts of good beer, not more than 300 c.c. or $\frac{1}{2}$ pint a day. Whenever the hydrochloric acid is diminished, the lighter meat varieties, chicken, pigeon, birds and fish should be allowed only, but a larger amount of carbohydrates conceded.

Constipation is a serious cause and constant accompaniment of gastric atony; it must therefore receive the most undivided attention. Purgatives should be used only as a last resort and the main reliance placed on diet. A pint of cold water in the morning on an empty stomach, black or graham bread, abundance of vegetables—turnips, carrots, asparagus, tomatoes, rhubarb plant, beans, peas, lentils, noodles, macaroni, barley, sweet compots, plums, figs, apples, currents, cranberries, cider. Sweeten everything with milk, sugar, butter-milk, kefir, sourmilk, honey. An abundance of these articles very rarely fails to bring about regular passages without medicines. Whenever a drug is positively unavoidable, I prefer cascara sagrada. In pronounced atony, constipation can not be treated by this diet only, because it increases the weight of ingesta.

The hydropathic treatment consists in cold morning sponge baths, cold wet packs and *Priessnitz* bandages to epigastrium. I am in the habit of ordering a daily bath in severe neurasthenic myasthenia which contains 3% chloride of sodium and 2% sodium bicarbonate;—temperature of bath 98°;—to remain in 20 minutes.

ELECTRIC TREATMENT:—

Intragastric, with the Einhorn electrode (Hemmeter's modification) in the stomach; the faradic current is applied up and down over the spinal column and over the abdominal muscles. The constant current is applied in the same manner in the strength of 20 milliamperes and for about 10 minutes Systematic massage both general and local over the stomach is an important adjuvant.

MEDICINAL:—

This form of treatment should be as limited as possible. The most approved tonic for the motor function is strychnin.

R

Strychnin Sulphatis gr. $\frac{1}{3}$,

Elixir gentianæ cum Ferri chloride, ʒvi M.fl. ʒss t.i.d.

Where the hydrochloric acid is deficient it must be supplied where it is excessive it must be neutralized by the following;—

R

Magnes. ust. 15.0

Bismuth Carbonic.

Natron bicarbonat. a.a. 5.0

Extract Strychni 0.1. M.

$\frac{1}{2}$ teaspoonful one half an hour after meals. Creosote and orexin are claimed by competent authorities (Pick and Penzoldt) to be able to excite the peristalsis; the latter may be used where there is an or subacidity.

LAVAGE:—

As a rule one will be able to get along without lavage in the first stage of motor insufficiency. But where the food remains in persistently over time, I have seen improvement of muscular tonicity follow the rapidly alternating cold and warm intragastric douche; this exerts a powerful and stimulating effect also on the secretion when it is defective, when the latter is excessive the douching should be carried out with alkaline water.

TREATMENT OF MOTOR INSUFFICIENCY OF THE SECOND DEGREE — FULLY DEVELOPED DILATATION.

This may be considered under two headings; — (1) Medical, (2) Surgical.

THE DIET is essentially based on the same principles as in simple myasthenia; the amount of permissible liquid must not exceed 1500 c.c. in 24 hours. With exaggerated vomiting and pains, I would recommend exclusive feeding by the rectum for 14 days. A specified diet list for both simple atony and pronounced dilatation will be appended. It is impossible to treat the latter form successfully without lavage, this is not only a palliative measure of great value, but in cases of atonic dilatation due to muscular weak-

ness and not dependent upon mechanical obstruction, it may even be able to effect a cure when combined with other means, presently to be described.

The first washings are carried out with pure warm water, but the last ones are done with solutions adapted to the chemical and septic state present in the organ. For instance, if there is great excess of hydrochloric acid and fermentation by *sarcinæ* and yeast, Sodium biborate or bicarbonate should be added, as these are not only antiaacid but with regard to these organized ferments, antiseptic. If there is butyric or lactic acid fermentation, Boracic acid, 3%—Salicylic acid, 0.3%—Creolin or Lysol 10 – 15 drops to a quart should be used: But the stagnation can not be prevented from recurring by these means unless the motility is improved by other treatment.

ELECTRICITY should be employed externally and internally as described in previous chapter.

MASSAGE undoubtedly improves the gastric musculature but should only be used on days when the stomach has been washed out, because the mechanical compression may force stagnating masses into the intestines, thus spreading the putrefaction. Abdominal bandages properly adapted and applied have proven a valuable palliative measure. Hpdrotherapeutic applications are indispensable and should be used as described in the last paragraph.

MEDICINAL TREATMENT has a twofold object; (1) To promote the motor function, (2) To prevent as far as is possible, gastric fermentation and decomposition. The only drug in which I have any faith for improving gastric peristalsis is strychnin sulphate, it should be given in heavy doses, not less than 1–30 grain for adults, t.i.d.

Boas combines strychnin with an antifermentative in the following manner;—

R	Extract strychnin	
	Codein phosphoric.	a.a. 0.03
	Bismuth salicylici basic	0.5
Sig.		M.

Make into 20 powders, one powder after each meal.

F. Kuhn has proposed salicylic acid 0.5 gm. per dose—Salicylate of sodium, 15 – 30 grains—Saccharin and Sodium benzoate, of

each form 10 – 30 grains per dose to counteract gastric fermentation. Carbolic acid was first used by Naunyn for the same purpose. When there is marked lactic or butyric acid fermentation, there is not a better agent than hydrochloric acid to counteract it:— 20 – 30 drops of the dilute form in ℥ii water, through a glass tube. Salol, Naphthol, Beta Naphthol Bismuth and Beta Naphthol Bismuth benzoate or Benzonaphthol. The French are very enthusiastic concerning antifermentative treatment of gastrectasia, but it is certain that this treatment alone with lavage and proper diet is fallacious.

Dujardin Beaumetz employs;—

R

Bismuth salicyl. Magnes. Usta. Sod. bicarb. a.a. 10 grm.

Sig.

M.

To be divided into 30 powders, one powder after meals. My own formula for gastric fermentation particularly when associated with putrid Diarrhœa is;—

R

Beta Naphthol. Bismuth. benzoatis	℥ii
Bismuth Salicylatis	℥ii
Magnesiae Usta	℥ii
Saccharin	℥ii
Menthol	℥ii

Sig.

M.

To be divided either into 12 or 24 powders to suit the indications; if there is much fermentation, it should be divided into 12 powders and one given 4 times daily. Otherwise it should be divided into 24 powders and one given every 3 hours.

For vomiting, lavage is the most efficacious treatment but if it fails a hypodermic injection of morphine and atropiæ sulphat will be called for. As a rule menthol and chloroform do not dissappoint when used for the relief of vomiting. The following formula is practical;—

R

Mentholi,	gr. xvi
Chloroform,	gtt. xxiv
Elixir simplic,	q.s. fl. ℥ii

Sig.

M.

fl. ℥ii every hour. Insomnia must sometimes be treated as

these patients imperatively need rest for this purpose Chloral gr. xv per enema is most advisable.

SURGICAL TREATMENT:—

The operations that have been suggested for the relief of Motor insufficiency vary according to the object to be accomplished. Motor insufficiency from simple atonic dilatation may be relieved by reducing the size of the stomach by excision of a piece of the same, an operation known as *Gastrorrhaphy*.

If the Pylorus is stenosed by a simple cicatrix or hyperplastic sphincter Loretas *Digital Divulsion* of the *pylorus* is an operation which judging from the statistics is an unsafe and unreliable procedure. The *Pyloroplastic Operation of von Heinecke — Milkulicz*, which Boas terms the ideal surgery for the relief of pyloric stenosis of a benign nature produces more permanent results.

There are two more, *Gastroenterostomy* and *Resection* of the pylorus. The Indications for these operations and their technique are subjects concerning which the reader must be referred to textbooks on surgery. The larger portion of dilatations is undoubtedly due to some obstacle to the exit of the chyme (is chochymia as Einhorn calls it) and it is rational to presume that purely medical means can not effect a permanent cure of these conditions. But the obstructions or obstacles to the chyme are not even all found in the stomach itself, for in the account given under the etiology, distended gall bladder, gall stones impacted in the diverticulum of Vater, floating kidney, duodenal cicatrices and neoplasm peritoneal adhesions etc., have been referred to, and all of these give their separate and distinct indications for operation.

DIET FOR MOTOR INSUFFICIENCY OF THE FIRST DEGREE

—ATONY—MYASTHENIA.

At 8 A.M.	Milk 100 grms. Toast + 30 gr. Butter	= 401.2 Cal.
At 10 A.M.	50 gr. Wheat bread + 30 gr. Butter + 60gr. scraped beef	= 415.2 Cal.
At 12 M.	150 gr. boiled Beef. + 50 gr. potatoe purée or macarony	= 439.3 Cal.
At 3 P.M.	100 gr. milk + 50 gr. Zwieback,	= 401.2 Cal.
At 7 P.M.	100 gr. Cold ham + 150 gr. wheat bread + 30 gr. butter	= 557.5 Cal.
	Total	2214.4 Cal.

About 3oz. good Portwine or Claret may be allowed during the day.

DIET FOR MOTOR INSUFFICIENCY OF THE SECOND DEGREE

—PYLORIC STENOSIS — MYASTHENIC DILATATION.

At 8 A.M.	50 gr. tea with 50 gr, milk sweetened with Saccharin, no sugar + 50 gr. toast	= 195.5 Cal.
At 10 A.M.	100 gr. scraped lean beef	= 437.0 Cal.
	30 gr. toast	= 77.7 Cal.
	10 gr. Butter	= 71.3 Cal.
	Total	586.0 Cal.
At 12 M.	150 gr. Roast beef	= 320.7 Cal.
	50 gr. potatoe purée	= 63.7 Cal.
	Total	384.4 Cal.

In place of the potatoe purée the same of spinach, carrots, peas or beans may be allowed in the same quantity.

At 2 P.M.	50 gr. Cream	= 107.3 Cal.
At 4 P.M.	100 gr. Tea or coffee with Milk, no sugar, but saccharin 50 gr. Toast	= 195.5 Cal.

At 7 P.M 100 gr. broiled white	
	or yellow perch or oysters = 71.75 Cal.
50 gr. Wheat bread	= 129.0 Cal.
10 gr. Butter	= 71.3 Cal.
100 gr. Cream	= 214.0 Cal.
At 9 P.M. 50 gr. Cream	= 162.3 Cal.
	<hr/>
	Total 1885.15 Cal.

In atony and dilatation as well as in carcinoma, experience is the best guide for enlarging and varying the diet. Every new article of diet must at first be tried with great caution; If liquids are well tolerated they may be increased and soups allowed for the noon meal. The daily lavage should at times be undertaken at hours when a test meal can be secured thereby which will incidentally instruct the physician concerning the digestibility of new foods and what is more important the state of the motor function.

PROFESSOR J. C. HEMMETER'S
Synopsis or Scheme for examining Stomach Patients at
THE MARYLAND GENERAL HOSPITAL.

Medical No.... Name..... Address..... Age..... Color.....
Sex..... Social Condition..... Diagnosis..... Date.....

HEREDITARY FACTS OF IMPORTANCE.

PREVIOUS HISTORY—Severe Constitutional Diseases.

First appearance of symptoms and cause? Did they appear suddenly? Intenrally?
Or gradually? Continuous? Or remittent? What intervals? Occupation?
Habits? Alcoholism? Tobacco? Cold? Change of climate? Mental strain?
Trauma? Malaria? Did it begin with or without a chill? Fever? Yellow fever?
Constipation? Diarrhœa? Dysentery? Typhoid fever?

PRESENT HISTORY—Diseases of other organs. Dyseptic symptoms? Pressure?
Local and subjective complaints? Fullness? Pain? Distention? Restlessness?
Sounds in the digestive tract? Bowel movements? Nausea? Eructation?
Vomiting? Hæmatemesis? Appetite? Taste? Thirst?

LOCAL SUBJECTIVE SYMPTOMS. Any difficulty or pain on deglutition? If so, its reg-
ularity? Irregularity? Intensity? Duration? Effect of food on pain?
Do they occur in every position of body? or only in certain positions?
Time of onset after meals? Pain at night? On an empty stomach? Improved by
eating? Exaggerated by eating? Is pain diffuse? or circumscribed?

ERUCTATION—Duration? Occurring on full? or empty stomach? Is gas tasteless?
Odorless? Acid? Decomposed? After what foods? Presence of pyrosis or heartburn?

NAUSEA AND VOMITING—Occurs on full or empty stomach? Frequency? Taste of vomit?
Appearance of matter? Food particles? Proteids? Starches? Mucous? Bile?
Blood? Food eaten several days before? Does emesis relieve symptoms?

APPETITE AND THIRST. Accustomed diet? Mode of life? Anorexia? Bulimia?
Aversion to meat? Thirst?

BOWELS—Constipation? Diarrhœa? Undigested particles of food? Mucous?
Pus? Blood and source? Results of blood examination.

GENERAL NUTRITION. Emaciation? Loss of weight in pounds? In what time?

PHYSICAL EXAMINATION.

INSPECTION. Change in form of abdomen? Tumor? Gastric or intestinal peristalsis?

PALPATION—Time of examination? Temperature? Outline of stomach? Upper border?
Lower border? Presence of tumor? Movement of tumor? Was stomach full or
empty? Pain on pressure? Diffuse or circumscribed? Succussion sound?

PERCUSSION—Limits of the stomach?

DISTENSION WITH AIR OR GAS—Limits of stomach? Results with intragastric bag?
Does tumor move with distension? Made more or less distinct?

ELECTRO DIAPHANY—Limits of stomach. Tumor?

EXAMINATION OF TEST MEALS.

Double test meal of the Md. Gen. Hos. at.....
Ewald breakfast at..... Contents drawn at..... Date.....

MICROSCOPICAL EXAMINATION. Quantity? Color? Odor? Food particles?
Froth or gas? Mucous? Bile? Blood?

CHEMICAL EXAMINATION. Reaction? Free Acid? Free HCL? Lactic acid?
Amount free HCL? Combined HCL? Amount acid salts and organic acids?
Total acidity? Erythro dextrin? Deficit of HCL?

PEPSIN.

Albumin digested in pure filtrate in...min. Albumin digested in acidified filtrate in...min.
Albumin digested in HCL and pepsin filtrate in...minutes.

RENNET.

Milk coagulated by rennet in.....min. Milk coagulated by rennet zymogen in.....min.
Rennet zymogen active in dilution 1: Contents after meal previous evening at 8 a.m.
Contents after lavage previous evening at 8 a.m. Time of salol reaction.....minutes.
Time of iodide of potassium resorption test.....minutes.

MICROSCOPICAL EXAMINATION. Bits of tissue? Bacilli? Yeast cells? Bits of mucosa?

URINE. Amount? Urea? Reaction? Indican? Performed sulphates? Albumen?
Tube casts? Ethereal sulphates? Ratio? Sugar? Specific gravity?

TREATMENT.

Diet? Medicines? Electricity? Massage? Hydrotherapy? Lavage? Results.

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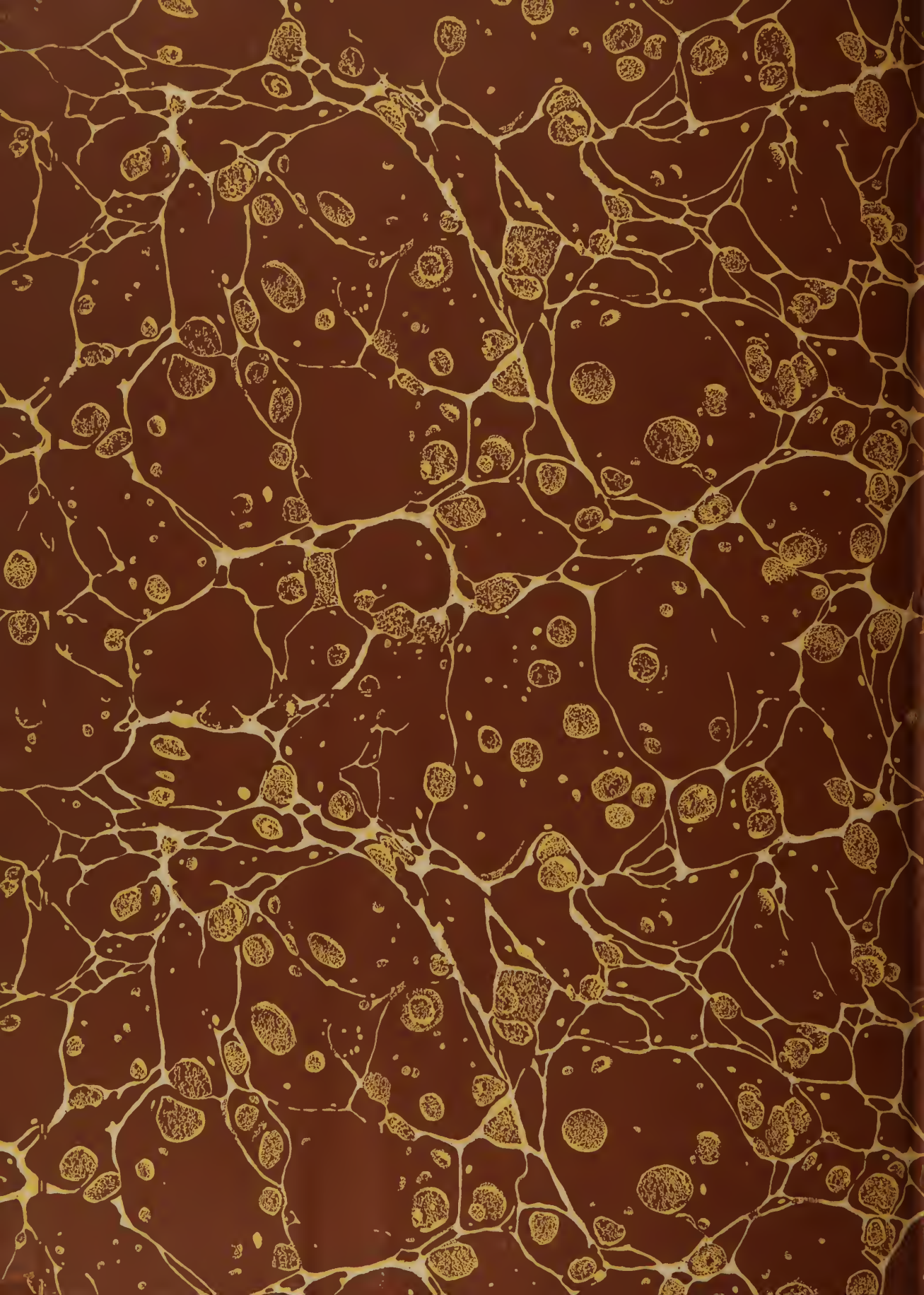
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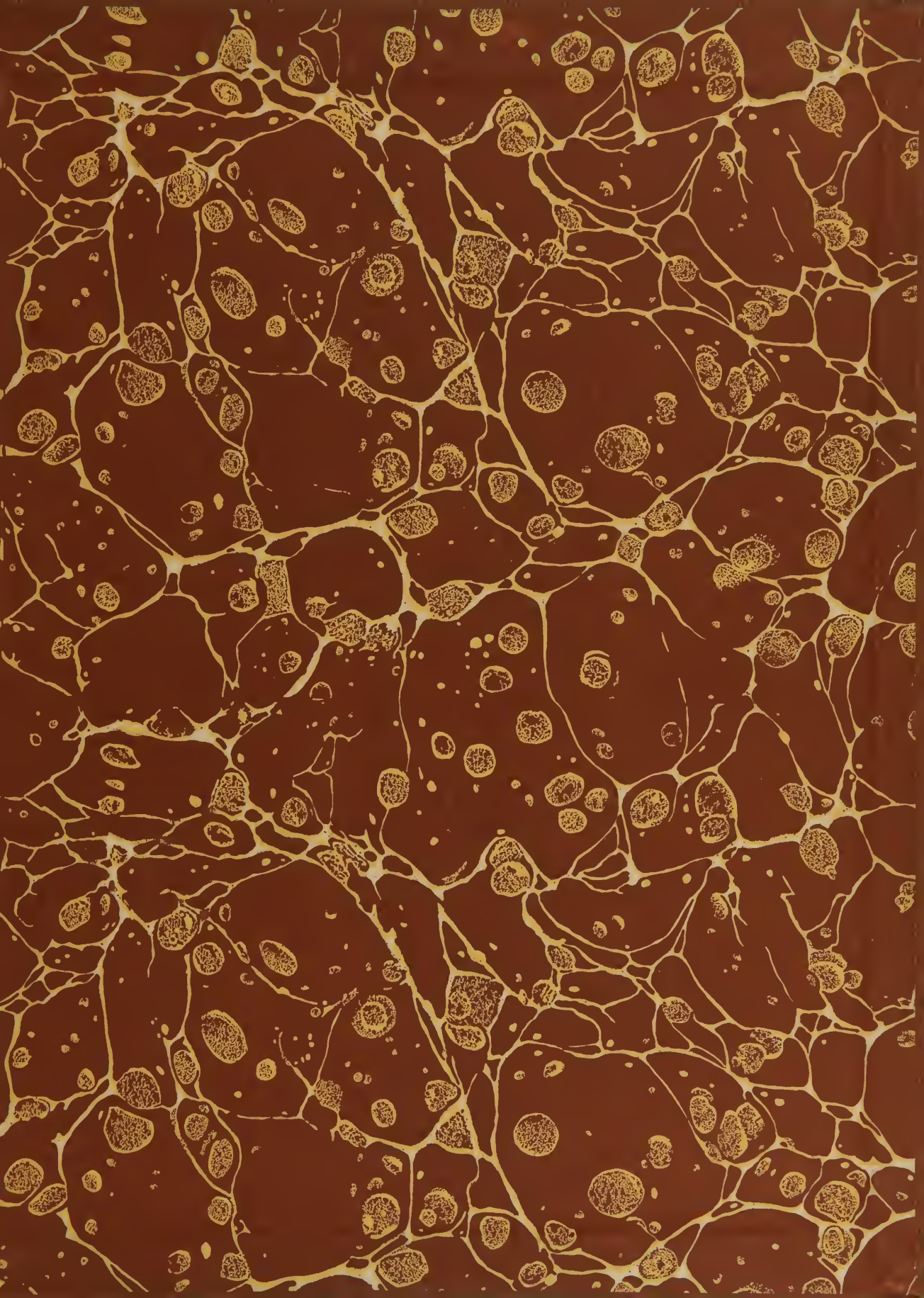
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